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INTRACRANIAL ANEURYSMS.[1]

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(From the Walter and Eliza Hall Institute of Research in Medicine and Pathology.)

AND when the child was grown, it fell on a day, that he went out to his father to the reapers.

And he said unto his father, My head, my head. And he said to a lad, Carry him to his mother.

And when he had taken him, and brought him to his mother, he sat on her knees till noon, and *then* died.

And when Elisha was come into the house, behold, the child was dead, *and* laid upon his bed.

He went in therefore, and shut the door upon them twain, and prayed unto the LORD.

And he went up, and lay upon the child, and put his mouth upon his mouth, and his eyes upon his eyes, and his hands upon his hands: and he stretched himself upon the child; and the flesh of the child waxed warm.

Then he returned, and walked in the house to and fro; and went up and stretched himself upon him: and the child sneezed seven times, and the child opened his eyes.[1]

II Kings, Chapter IV, Verses 18, 19, 20, 32, 33, 34, 35.

Search of the records of the Royal Melbourne Hospital prior to 1929 fails to reveal the diagnosis of intracranial aneurysm. Yet the subject is not a new one: there is a summary of contemporary

[1] The record of a clinico-pathological demonstration given at a meeting of the Victorian Branch of the British Medical Association held at the Royal Melbourne Hospital on May 20, 1936.

[1] This interpretation of the illness of the Shunammite woman's son is at variance with that put forward by Osler and McCrae ("Principles and Practice of Medicine", ninth edition, page 380). In dealing with sunstroke it is stated: "It is one of the oldest of recognizable diseases. The case of the son of the Shunammite woman is perhaps the oldest on record."

knowledge of the subject, surprisingly detailed, in Gowers's "Diseases of the Nervous System", published in 1888. The signs of aneurysm of the various intracranial arteries are described and the symptoms and signs of rupture are clearly delineated, although it was probable that the diagnosis was rarely made before death, and then only after Quinke had introduced the procedure of lumbar puncture in 1891. Of the congenital origin of so many of these aneurysms he was unaware, ascribing their causation to degenerative diseases of the arteries, trauma, embolism and syphilis. Undoubtedly the last mentioned played a larger part in the ætiology then than now, explaining in 144 cases collected by Gowers[1] in 1893 the high incidence upon the basilar artery (basilar artery, 41 cases; middle cerebral, 44; internal carotid, 23; anterior cerebral, 14; posterior communicating, 8; anterior communicating, 8; vertebral, 7; posterior cerebral, 6; inferior cerebellar, 3).

There is little need to describe the symptoms of rupture in detail. In broad review of 31 patients whose condition was diagnosed at the Royal Melbourne Hospital since 1929, it may be said that the onset occurs during very varying conditions, sometimes during bodily exertion, sometimes during sleep. One patient awoke from sleep screaming: "Oh, my head, my head", and then lost consciousness; another sneezed and immediately felt very severe headache; a boy fell from a cycle, and another patient fell and struck her head for a reason which she could not state. Several patients were found unconscious.

Most patients complained at or soon after the onset of very severe headache, sometimes generalized, more frequently frontal, temporo-parietal or occipital in location. Later the headache was usually felt over the occipital region with pains radiating down the back of the neck. Two patients felt as though something had burst inside their heads. Vomiting, giddiness, drowsiness and loss of power in the limbs were other symptoms described. Dr. R. J. Wright-Smith informs me that he has found 14 ruptured congenital aneurysms in a total of 1,351 autopsies performed for the coroner since 1930, bespeaking the rapid death which may occur within a few hours in this condition.

Sometimes death followed rapidly, within twenty-four hours; more frequently, after an initial somewhat brief period of coma, the patient's state of consciousness varied: he was irritable, resented interference, complained bitterly of headache at times, at other times was drowsy and lapsed into coma. The temperature was frequently elevated, rarely, in uncomplicated cases, beyond 38·3° C. (101° F.). The pupils were frequently dilated and unresponsive to light. The state of the deep reflexes varied; sometimes they were hyperactive, sometimes absent. The superficial abdominal reflexes were sometimes absent and the plantar responses sometimes extensor. Hemiplegic signs were common. Kernig's sign and stiffness of the neck were the rule, at any rate some hours after the onset. The cerebro-spinal fluid was thickly and evenly blood-stained. Albumin was present in the urine in three patients, sugar in one.

The ages ranged from sixteen to fifty-five years, 11 being below thirty years. In several there was a long history of attacks of headache and vomiting. Fourteen patients survived, seventeen perished, two after having recovered from a previous rupture. Fresh hæmorrhage frequently preceded death, bronchopneumonia being a contributory cause in two patients. One patient had arteriosclerosis, secondary to chronic glomerulonephritis. Polycystic kidneys were found at autopsy in one patient, and another had marked generalized atheroma, having previously had symptoms suggesting coronary occlusion.

Congenital aneurysms tend to develop at the point of bifurcation of the larger cerebral vessels on or near the circle of Willis. Of those definitely located at autopsy, six were upon the middle cerebral artery, three near its origin and three deep in the lateral sulcus. Three were on the junction of the anterior communicating artery with the anterior cerebral artery, and two were situated more peripherally on the anterior cerebral artery. Three arose at the obtuse angle formed by the internal carotid artery and the posterior communicating artery. As would be expected, it is those aneurysms of the middle cerebral artery embedded in the lateral sulcus and of the anterior cerebral arteries between the frontal lobes which most frequently rupture into the brain substance, the blood tearing the cerebral tissue and eventually reaching the lateral ventricles. In one such case the cerebro-spinal fluid contained only a few red blood cells four days after the onset; the next day, preceding death, the fluid was heavily blood-stained. The aneurysm was on the left middle cerebral artery, the left temporal lobe was ploughed up, each cerebral ventricle contained clot, and there were numerous small hæmorrhages in the brain stem, chiefly related to the floor of the fourth ventricle. In several cases the subdural space contained blood, owing undoubtedly to a tear in the arachnoid membrane.

In 1923, Harvey Cushing,[2] writing on the subject of intracranial aneurysm, stated: "How it is that a surgeon comes to write a note upon a lesion having such remote surgical bearings may be told . . . ." Yet this condition has since been treated surgically in chosen cases with success, Norman Dott being the chief protagonist of the method. Yet again Sir William Gowers has a surprise for us; he writes in the article mentioned above:

> The third method of promoting coagulation in the sac is to ligature the artery from which the diseased vessel derives its supply of blood. Hitherto this has only been adopted in the case of aneurysm of the internal carotid, for which the common carotid has been tied with success.

Nattrass[3] mentions a patient with an aneurysm of the internal carotid artery in whom ligature of

the artery was performed by Professor Grey Turner in 1928 and who was alive five years later. Dott[4] introduced the operation of direct application of muscle to the bleeding point in order to induce clotting and subsequent repair by fibrosis. Later, for the exact localization of these lesions, he commenced to utilize the method of angiography, introduced by Moniz, consisting of X ray photography of the skull during the intracranial circulation of radio-opaque substance following its injection into the internal carotid artery. If the aneurysm is upon the internal carotid artery, he suggests immediate ligature of that vessel in the neck. If the aneurysm is on or distal to the circle of Willis, and if conservative treatment appears likely to fail, he suggests the application of muscle to the tear. Certainly without operation the mortality is high.

On July 13, 1935, T.S., a male, aged thirty-five years, retired for the night, complaining of pain in the back, near the left shoulder blade. Soon afterwards his wife found him unconscious, twitching in the right hand. In between such attacks, which were repeated several times during the night, he was drowsy, forgetful of the events of the previous twenty-four hours, and complained of headache, pain in the neck and in the left scapular region on bending forwards. The next day his doctor reported weakness of the left arm and leg; the left plantar response was strongly extensor and there was sensory loss to pinprick and heat and cold over the left side of the body. He complained of headache and was confused mentally during the next two weeks.

From that time onwards he complained of occipital headache, increased by lying down, sometimes keeping him awake at night, and relieved by sitting up. At times he felt as though he were about to faint, and, when rising or stooping, experienced giddiness. Rarely his eyesight would seem blurred for prolonged periods, and in January, 1936, pain radiating down the arm from the shoulder became troublesome, induced by flexion of the head and coughing, and was worse at night.

His illness was investigated thirteen weeks after its onset; there was slight left-sided hemiparesis with some hypotonicity and wasting. The left-sided tendon reflexes were diminished, the left superficial abdominal reflexes were less active than the right, and the left plantar response was equivocal. The only defect in sensation was a diminution of appreciation of position and passive movement in the left great toe.

Lumbar puncture revealed an initial pressure of 150 millimetres of cerebro-spinal fluid. Response to jugular compression was normal. The cerebro-spinal fluid was clear and colourless; it contained one lymphocyte per cubic millimetre and had a total protein content of 0·015%; there was no increase in globulin. The colloidal gold test revealed no change from normal. The Wassermann test gave no reaction in cerebro-spinal fluid and blood. The urine was normal chemically and microscopically. The blood urea was 30 milligrammes per 100 cubic centimetres. X ray examination of the skull showed no abnormality, the calcified pineal gland being in the midline. X ray examination of the cervical spine showed localized spondylitis on adjacent borders of the fifth, sixth and seventh cervical vertebræ.

Encephalography was performed on November 28, 1935. Fifteen cubic centimetres of air were introduced without discomfort to the patient. The lateral and third ventricles were normal in size and shape and were symmetrically disposed in relation to the middle line.

The patient continued to complain of the symptoms described above; indeed, he said that he was unable to work, and implored that something should be done for him. The question of angiography was discussed and he agreed that it should be done. A small aneurysmal sac was visualized on the left side, between the internal carotid and anterior cerebral arteries (Figure I). This did not, however, solve the problem of treatment. The patient chose to avoid operation and he has since been able to carry on, relatively free from symptoms. His wife has instructions to bring him to hospital immediately should any untoward event occur.

Other difficulties in surgical treatment of such patients are illustrated by the following case.

T.G., a male of thirty-one years, was admitted to the Royal Melbourne Hospital under the care of Dr. Hume Turnbull. His wife stated that he had collapsed under a cold shower three hours before admission. His temperature was 35·6° C. (96° F.), pulse rate 65, and respiratory rate 24. His systolic blood pressure was 170 and diastolic pressure 120 millimetres of mercury. His arms were in tonic flexion, the lower limbs in extension, with feet plantar-flexed and inverted. The tendon reflexes were equally active on either side, the right-sided superficial abdominal reflexes were absent, and the right plantar response was equivocal and later extensor. The left-sided superficial reflexes were normal. There was a subhyaloid hæmorrhage, a large bean-shaped collection of blood with convex lower margin, below the right disk. The urine contained a large amount of sugar. Lumbar puncture revealed blood-stained fluid under a pressure of 250 millimetres.

Angiography revealed no abnormality of the left internal carotid and middle cerebral arteries, so the patient was returned to bed. He died four days later from a recurrence of hæmorrhage. At autopsy a small aneurysm, which had ruptured, was found growing from the obtuse

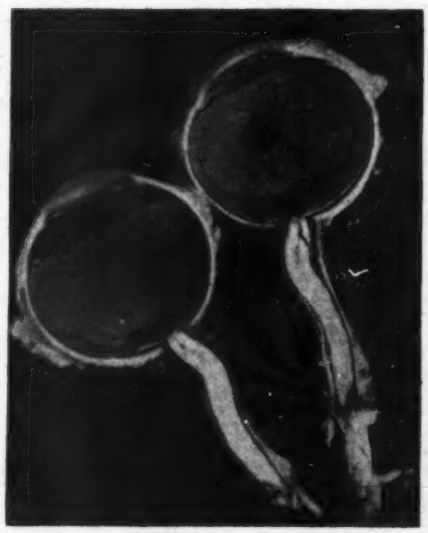


FIGURE III.

Section of the eye of T.G., showing the subhyaloid hæmorrhage and the pia-arachnoid sheath of the optic nerve distended with blood. As a *post mortem* change some blood has gravitated to the most dependent part of the eye.

angle of junction of the right internal carotid and posterior communicating arteries. The latter artery was much larger than that on the left side. There was a collection of blood clot, thickest at the base, on the right side and over the right hemisphere, penetrating deeply into the sulci, throughout the subarachnoid space. There was a small amount of recent clot in the fourth ventricle. The aneurysm is shown in low power magnification in Figure II. Section of the right eye (Figure III) revealed blood in the subarachnoid space reaching to the posterior pole of the eye. The hæmorrhage is probably

due to seepage of blood through the *lamina cribrosa*. The subhyaloid hæmorrhage has a very different appearance from those surrounding the disk when there is obstruction to venous return. The lungs showed gross œdema and patches of hæmorrhage.

Why the very definite alteration in the superficial reflexes was so misleading cannot be explained. It serves, however, as a warning. The patient's condition did not permit of injection on the opposite side, nor was the anterior cerebral artery on the left side visualized. Injection experiments, combined with X ray examination after death, made it doubtful whether the aneurysm would have shown in radiograms, so small was it, unless perhaps both lateral and antero-posterior views were taken. I have seen an aneurysm upon the anterior communicating artery displayed at *post mortem* examination, yet missed by angiography.

One other recent case may be mentioned.

S.C., a male, aged sixty-one years, was admitted to the Royal Melbourne Hospital on May 31, 1936, under the care of Dr. S. V. Sewell. In 1930 the diagnosis of coronary occlusion was made upon a history of constricting pain in the precordium, over the left shoulder and down the outer aspect of the arm to the elbow. The electrocardiogram showed changes consistent with coronary arteriosclerosis. Similar attacks continued, and in March, 1936, he complained of severe pain over the left eye, occurring at first chiefly in the morning, later lasting all day. He commenced to sleep badly and during the week previous to admission he vomited repeatedly, became drowsy and failed to recognize his relatives. Examination, in brief summary, revealed a large inactive left pupil with defective adduction and elevation of the left eye. The deep reflexes were very active, the left-sided abdominal reflexes were absent and the plantar responses were extensor. The apex beat was in the fifth intercostal space, 8·75 centimetres (three and a half inches) from the mid-line. There was a blowing systolic murmur at all areas. The systolic blood pressure was 184 and the diastolic pressure 110 millimetres of mercury. The peripheral arteries were thickened and tortuous and the retinal arteries showed some variation in calibre.

Lumbar puncture revealed evenly blood-stained cerebrospinal fluid. The patient died after transient improvement. Lateral to a large left posterior communicating artery there was a mass of blood clot which issued from a ruptured aneurysm (Figure IV) at the point of origin of the above-named vessel. On removal of the clot the aperture was seen to be as large as the orifice of the incised internal carotid. He would be brave who would disturb such a clot.

The vessels showed gross atheroma. It is probable, however, that the aneurysm was of congenital origin, because microscopic examination of the points of bifurcation of other cerebral vessels showed the typical defect of the muscular coat, to be referred to later, which predisposes to the dilatation at these points (Figure V). A high blood pressure and narrowed peripheral vessels would increase the stress upon such places of diminished resistance.

From the point of view of surgical treatment these are an unfortunate group of cases. Aneurysms of the internal carotid artery causing the "cavernous sinus syndrome" have been scarce, and it is in ligation of the internal carotid artery for such an aneurysm that surgery has most to offer. From published cases the risk of hemiplegia would appear to be a small one. The location of such an aneurysm may be obvious without angiography, as it tends to involve the cranial nerves in the cavernous sinus (third, fourth, sixth ophthalmic and maxillary divisions of the fifth nerve).

Unless the aneurysm be upon the internal carotid artery, I would believe that the adoption of conservative treatment, sedatives and repeated lumbar puncture, when necessary to reduce the intracranial pressure, is the best method of treating rupture, unless there are repeated fresh hæmorrhages and it is apparent that such treatment will fail. It is, however, difficult to be certain of this, and death often occurs with little warning. In some instances the aneurysms are multiple and cure of one rupture may be followed at a later date by hæmorrhage from another sac. However, as mentioned before, rupture brings with it a rather greater chance of death than of life; and if the percentage of deaths in a large series can be decreased by intracranial operation, the method is worth application. There

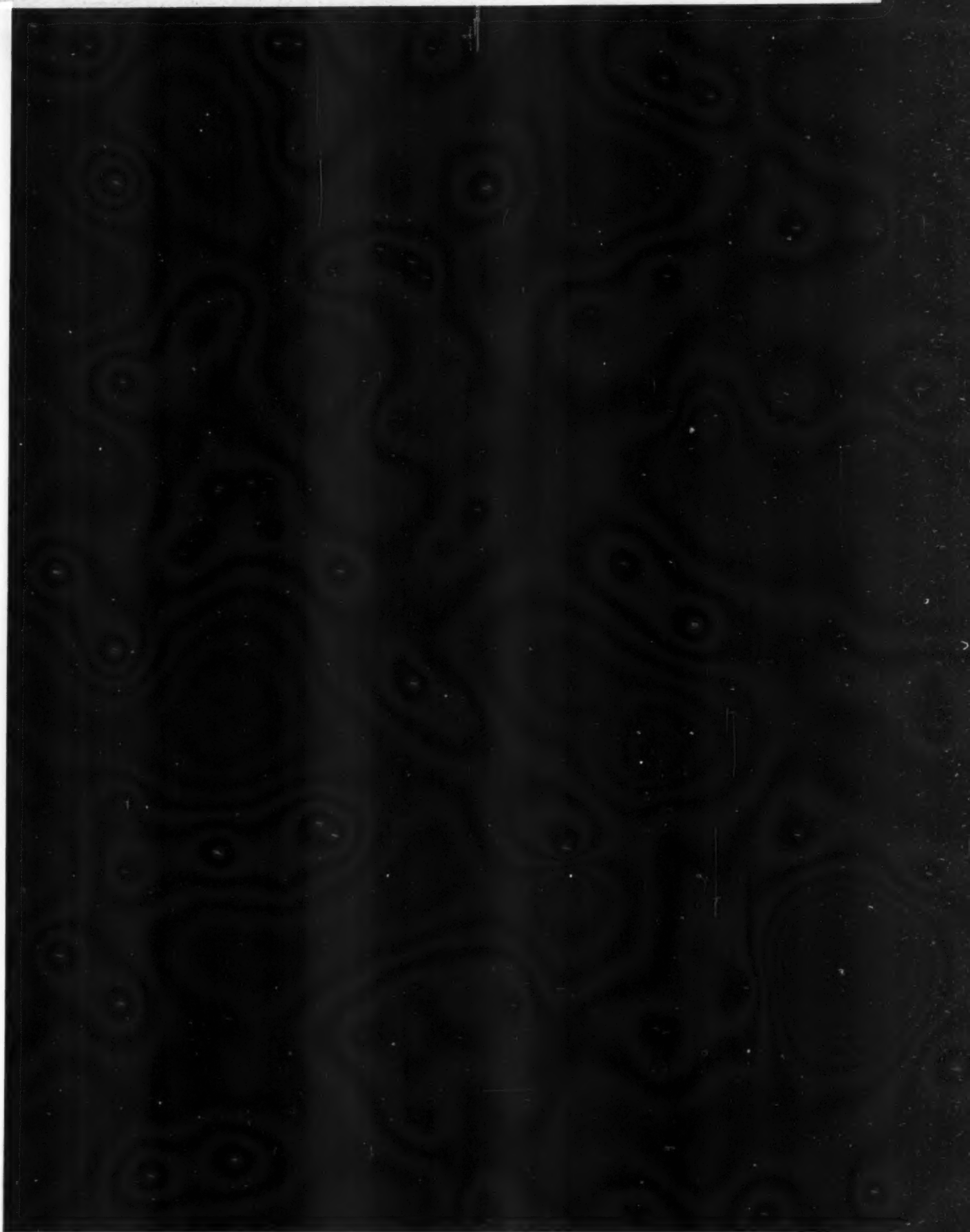
FIGURE IV.

Photograph of the base of the brain and the circle of Willis in the patient S.C., showing blood clot at the large aperture of an aneurysmal sac. Atheroma of the internal carotid artery is very obvious.

can, however, be no guarantee that a patient who would have lived without operation will survive such interference. These remarks are tentative, made in the course of a demonstration, and represent a temporary phase in the evaluation of a method as determined by recent experiences.

The following case has a direct bearing upon the problem under discussion.

In 1925 and again in 1930, M.M., aged twenty-nine years, suffered from two severe illnesses, diagnosed as meningitis and meningismus, which were similar to the two later attacks described in more detail. A few days before her admission to the Royal Melbourne Hospital, under the care of Dr. S. V. Sewell, in 1933, she had experienced the dramatic onset of pains over the whole of her head and down the back of the neck. She felt giddy and in an hour's time commenced to vomit. She remained in bed, vomiting and screaming, and the day after the onset pains were noticed in the back and along the back of the thighs. She was unable to bend her neck or legs.



ILLUSTRATIONS TO THE ARTICLE BY DR. GRAEME ROBERTSON.

FIGURE I.

Angiogram showing aneurysmal sac, filled with thorotrast, on the circle of Willis in the patient G.S.

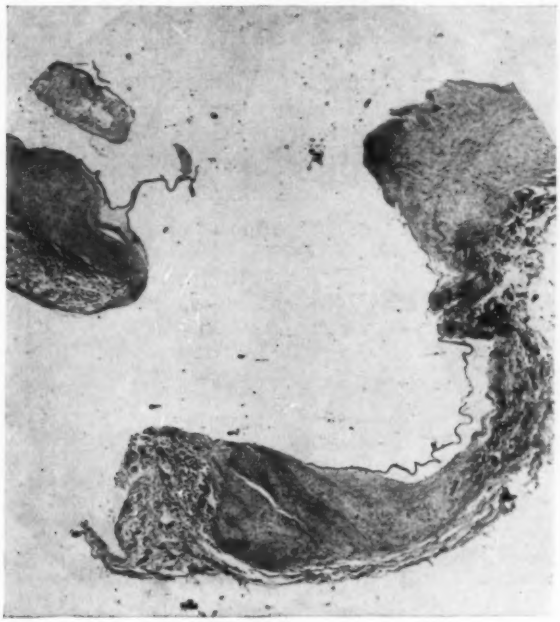


FIGURE II.

Photomicrograph of ruptured aneurysmal sac in the patient T.G. The fibrous nature of the wall of the sac and the absence of muscle and internal elastic lamina are well seen in sections stained with hæmatoxylin and Van Giesen's stain.

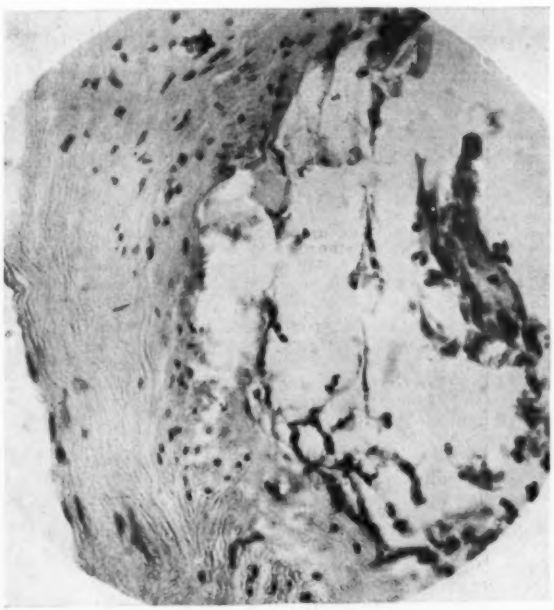


FIGURE V.

Photomicrograph of the point of bifurcation of a large branch of the middle cerebral artery in S.C. The muscular middle coat is seen to cease on each side, the actual point of bifurcation being bridged by connective tissue. The intima and adventitia appear to be normal.

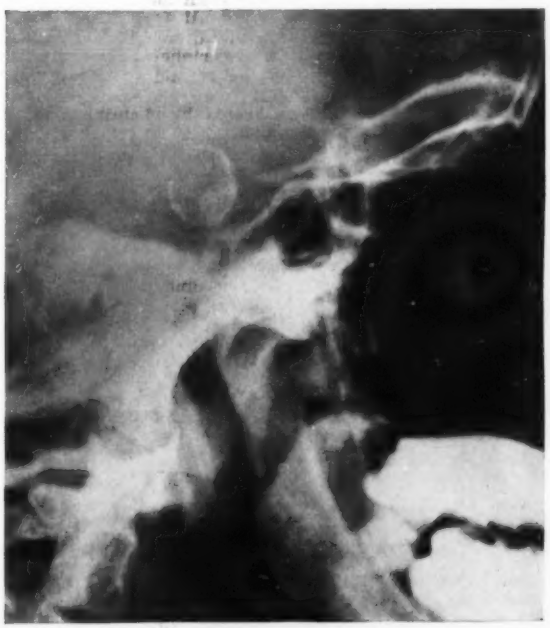


FIGURE VII.

X ray photograph of skull, showing calcification in an aneurysmal sac.

ILLUSTRATIONS TO THE ARTICLE BY DR. LEONARD B. COX AND I. M. McPHEE.

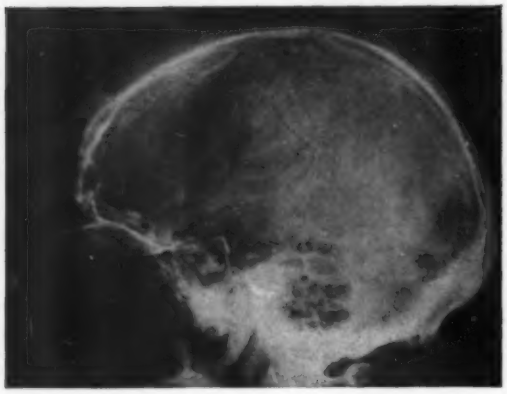


FIGURE II.
Case I, showing the enlarged pituitary fossa, with the neighbouring calcification and the rarefaction of the bones of the vault.

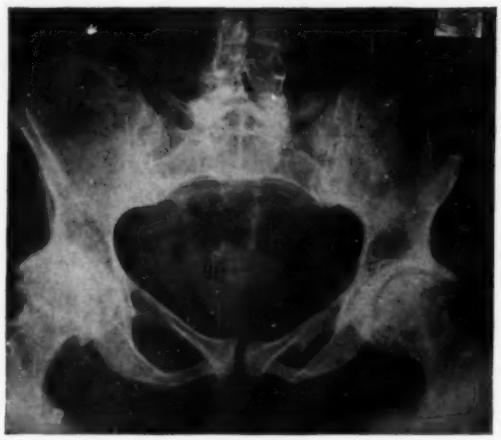


FIGURE III.
Case I, showing osteophytes and the general rarefaction of the bones.

ILLUSTRATIONS TO THE ARTICLE BY DR. E. H. GOULSTON.

FIGURE I, showing fracture.

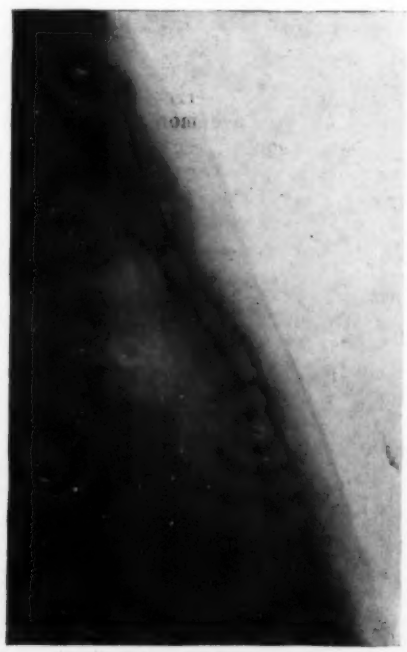


FIGURE II, showing end-result.

Examination revealed no abnormality except for the presence of Kernig's sign and stiffness of the neck. The cerebro-spinal fluid was deeply blood-stained. The patient gradually recovered and remained well until May 17, 1936, when she again complained of intense headache across the forehead and down the nose, which gradually became less during the ensuing days. On May 27 she suddenly cried: "Oh, my head", complained of intense headache, and rapidly lost consciousness. When examined two hours after the onset, the deep reflexes were very active, especially on the left side, the abdominal reflexes were absent, the right plantar response was flexor, the left equivocal. There was very great rigidity of the neck, and Kernig's sign was present on both sides. Blood-stained cerebro-spinal fluid, under an initial pressure of 280 millimetres, was removed at lumbar puncture.

In deciding whether arteriography should be performed, the fact that the patient had recovered from three previous hæmorrhages was taken into consideration. Surgical intervention would inevitably be blamed should she die following it. Beyond commenting upon the number of subarachnoid effusions, there was little reason to suspect that the diagnosis was other than that of ruptured congenital aneurysm. After an increase in the severity of headache the patient lapsed into coma and died two days after admission.

Post mortem examination revealed that the blood in the subarachnoid space was due to escape of blood from the fourth ventricle, each ventricle being filled with blood clot. The origin of the blood was from a small area in the wall of the lateral ventricle. The origin of the hæmorrhage was not apparent, but may be revealed in serial celloidin sections of the area.

In this paper a discussion of the pathological basis of such intracerebral hæmorrhages, interesting as they are, would be a digression. The case is mentioned because it draws attention to the fact that the clinical diagnosis of ruptured aneurysms of the circle of Willis usually depends upon the sudden effusion of blood into the subarachnoid space; there are other causes of such an effusion. It is of interest to mention in this connexion that Dr. Wright-Smith, in the series of autopsies mentioned above, in which fourteen congenital aneurysms were discovered, found in twelve cases diffuse subarachnoid hæmorrhage in the absence of a history of injury or a demonstrable aneurysm.

There has been some disagreement of opinion as to whether lumbar puncture should be repeated in such cases, owing to the fear that the lowering of intracranial pressure will increase the hæmorrhage. High intracranial pressure, however, may cause death, and if it is rising, as shown by slowing of the pulse, rising blood pressure and increasing coma, lumbar puncture should be performed, the pressure being lowered to about 140 millimetres of cerebro-spinal fluid. It is probable that the hæmorrhage is reduced more by lowering of blood pressure than it is increased by the reduction of intracranial pressure. The fluid should be drained very slowly after the absence of a pressure cone has been proved by the rapid response of the pressure in the lumbar sac to compression of the jugular veins.

The next specimen is an example of an aneurysm which produced signs only as a space-filling lesion.

M.P., a male, aged twenty-eight years, was admitted to the Royal Melbourne Hospital on February 3, 1936, under the care of Dr. R. P. McMeekin. Six months before admission he first noticed soreness behind both eyes, especially the left, associated with frequent frontal headache; these headaches were of the same type, but more frequent and severe than those from which he had suffered for ten years. In October, 1935, headache, which lasted for three weeks, commenced under the left temporal region and subsequently recurred intermittently. He felt severe pain at the back of the left eye and from there the pain radiated to a spot in the anterior part of the left temple, which seemed to throb. Early in November, 1935, he commenced to suffer from "smelling turns"; an acrid smell, which he could not adequately describe, commenced in the upper part of the left nostril and increased with each breath, merging into a feeling of dizziness. He felt as though something had flashed through his mind, "usually about a person I had known long ago, to whom I said something which I cannot remember". He afterwards wondered why he thought of that person, although he could not remember who it was and was unable to recapture the conversation in detail. After a moment it was all over and he felt "very bright". The attacks usually occurred each night when he was falling asleep and he was unable to sleep for some time afterwards. During the last week in November he had a severe attack in which consciousness was more impaired than usual. For an indefinite period he had suffered from cough with thick yellow expectoration, which was sometimes blood-stained.

Figure VI.

Aneurysm growing from a point of bifurcation of the middle cerebral artery and embedded in the uncinate region of the patient M.P. The inferior surface of the frontal lobe has been amputated.

Upon repeated examination the right-sided superficial abdominal reflexes were sometimes found to be diminished. There were no other signs, beyond generalized high-pitched rhonchi over his chest. The systolic blood pressure was 138 and the diastolic pressure 100 millimetres of mercury. Cerebro-spinal fluid was normal in November, 1935. On lumbar puncture the initial pressure was 190 millimetres of cerebro-spinal fluid. The cells were: 20 lymphocytes, 2 large mononuclear cells, 12 erythrocytes per cubic millimetre of fluid. There was no increase in globulin. The Wassermann test gave no reaction. X ray examination of the skull and of the chest revealed no abnormalities.

Of the localization of the lesion there could be no doubt; the description of the uncinate epilepsy could not be more clear. Suspicion of a tuberculoma was raised. In order to be certain that there was a space-filling lesion, and to lateralize it with certainty, preliminary encephalography was undertaken. Five cubic centimetres of air were injected and, perhaps owing to difficulty in insuring accurate posturing of the head because of voluminous dressings, no air entered the ventricles. Difficulty was experienced throughout anæsthesia (intratracheal administration of ether); the patient was cyanosed and enormous variations in blood pressure were noted. The brain on exposure was "pink" in colour, and the surgeon, Dr. A. E. Coates, palpated a hard mass, which he thought to be pulsating, in the region of the uncus. No attempt was made to remove it. The patient was extremely restless on return to the ward and died four hours later.

At autopsy, subjacent to the tip of the uncus there was a round, dark mass, eighteen millimetres (three-quarters of an inch) in diameter, with a fibrous wall containing laminated blood clot. Its anterior surface rested upon the posterior aspect of the middle cerebral artery, its neck communicating with the artery, where it gave off a small branch destined to penetrate the brain substance (Figure VI). There were a number of areas of hæmorrhage throughout the white matter of the *centrum ovale* and in the basal ganglia in the brain, in the epicardium and endocardium in the heart, and in the interlobar fissures in the lungs. These were evidently the result of the enormous variations in blood pressure.

Calcification within the periphery of an aneurysm may occur when clot lamination has commenced within it. The presence of an aneurysm may then be inferred from X ray films, a very characteristic ring-like shadow, Albl's ring, being evident. The writing of these words has been responsible for the recollection of a film seen seven years ago and the posthumous diagnosis of a lesion which was previously thought to be a neoplasm.

J.B. was first admitted to the Royal Melbourne Hospital in April, 1916, unconscious, breathing stertorously and bleeding from ears and nose. The deep reflexes were equally active, superficial reflexes were absent and the plantar responses were extensor. When the patient recovered consciousness he complained of severe headache, which lasted for three months; there was marked ptosis of the left upper lid, the left pupil was dilated and reacted sluggishly to light, and the left internal rectus muscle was paralysed. No investigations were performed. The diagnosis of fractured base of the skull was made.

The patient was readmitted on June 17, 1929, under the care of Sir Alan Newton. He complained that twelve years ago he had noticed spasmodic contracture of the toes of the right foot, and for this two toes were amputated. In 1926 the whole lower limb seemed stiff, and wasting commenced in its muscles. He stated that his left eye tended to swing outwards when he became excited. In 1928 he noticed spasmodic contracture of the flexors of the right elbow and stiffness of the fingers. There had been increasing deafness in the left ear for two years, and during six months he had noticed twinges of pain in the left side of the head. He complained of being "spasmodically tongue-tied".

On examination there was loss of upward movement of the left eye and rigidity of the right arm and leg without reflex or sensory change. In spite of the reported absence of reflex changes, a review of a film of the patient's gait, taken by Dr. Douglas Thomas, suggests that the motor state was at least partly due to involvement of the pyramidal tract, although the postures of the hand and foot were not typical. There was probably superadded extrapyramidal rigidity. The arm was held rigidly adducted, the elbow and wrist were strongly flexed, and the fingers were extended. The lower limb was rigidly extended at the knee and was swung forwards by circumduction during rotation of the pelvis. There was eversion at the hip joint and the foot was inverted and somewhat plantar-flexed. The X ray film showed a large ring of calcification lateral to the left posterior clinoid process (Figure VII).

In November, 1929, the tumour was exposed in this region, but owing to serious hæmorrhage the wound was closed. The patient was readmitted to hospital in 1930, able to say little but "yes" and "no" since the previous exploration and with a complete right-sided homonymous hemianopia. In May, 1930, the temporal lobe was reelevated to expose the floor of the middle cranial fossa. The tumour was found to be surrounded by a firm, hard capsule, the upper part of which was cut away and its contents coagulated and scraped out with a diathermy knife. Hæmorrhage was encountered while cleaning out its deeper part. The patient died seven days later of pneumonia.

Section showed "coagulated tissue and hæmorrhage" and on reexamination of the material it was seen that it may quite well have been laminated contents of an aneurysm. The *post mortem* report gave evidence of disintegration of the lower and middle parts of the temporal lobe, as though it had been deprived of blood supply. The base of the skull showed a roughened area in the medial part of the middle fossa, the tumour having apparently been completely removed.

It is possible that the initial onset in 1916 was due to a leak from the aneurysm, but the report of bleeding from ears and nose suggests that he had in reality sustained a fractured base of the skull, perhaps involving the left third cranial nerve. Within a few years right-sided symptoms indicating progressive involvement of the motor system appeared, partial palsy of the third cranial nerve remained, and he suffered from slight left-sided headache. These symptoms were due to a large aneurysm growing in the region of the circle of Willis and involving the left cerebral peduncle and perhaps the basal ganglia by pressure. It is possible that the aneurysm was traumatic in origin, due to injury to the internal carotid artery in the basal fracture. This case may with interest be compared with one described by Collier,[5] in which two circular shadows were seen in the supra-pituitary region. Sir Percy Sargent removed what proved to be one lobe of a clotted and calcified aneurysm. The patient died months later of pituitary cachexia, and the second shadow was found to represent the second lobe of an aneurysm of the anterior communicating artery.

The pathology of such aneurysms is of great interest. Eppinger[6] in 1887 suggested that many aneurysms of smaller arteries, like those of the circle of Willis, have a congenital basis in a defect of the elastic properties of the arterial wall. Wiley Forbus[7] demonstrated that in a considerable percentage of all persons there is, at the dividing point of arteries, a defect in the muscular coat, a gap bridged by connective tissue. This defect is shown in the photomicrograph of a bifurcation of the middle cerebral artery of the patient S.C. referred to above (Figure V). By suggesting that the independently formed muscular coats of adjoining vessels fail to fuse, Forbus correlates this with the developmental fact that each endothelial tube which joins up to form the vascular tree develops its own muscularis from surrounding mesenchyme. So at a

point which he showed experimentally to be subject to the greatest strain there is an area of diminished resistance and here the force of the blood stream may cause aneurysmal dilatation. When an aneurysm is present he showed this defect to be constant throughout the cerebral vessels, and in some cases in mesenteric and coronary vessels as well. This explains the fact that aneurysms may be multiple, and I have seen a case in which subarachnoid and retroperitoneal hæmorrhage coexisted. Aneurysms are more common in the cerebral than in the systemic vessels, because the former are large thin-walled vessels, bearing high pressures in spite of the double S-shaped bend of the carotid artery. Branches are placed, in some instances, so that the point of bifurcation is in the direction of thrust of the blood stream, the vessels are poorly supported, and sometimes the paired vessel in the circle of Willis is aplastic, subjecting its fellow to greater strain.

The wall of the aneurysm is composed only of connective tissue; no muscular tissue is found within the sac, and the internal elastic lamina always ceases close to its neck (Figure II).

An aneurysm due to any cause may, of course, rupture, and in this connexion it may be of value to record what was apparently dilatation of a vessel wall weakened by an arteriosclerotic process.

J.C., a male, thirty-one years of age, was admitted to the Royal Melbourne Hospital under the care of Dr. Konrad Hiller on March 12, 1930. The patient, a night-watchman, was found unconscious at work. He had previously consulted a doctor for transient fits of dizziness accompanied by blurring of vision, and was told that he had some degree of cardiac failure. On admission his systolic blood pressure was 157 and his diastolic pressure 70 millimetres of mercury. His urine was loaded with albumin and the heart was enlarged, the apex beat being in the sixth intercostal space. The left pupil was larger than the right, neither reacting to light. The tendon reflexes were more active on the left side and both plantars were extensor. Lumbar puncture revealed evenly blood-stained cerebro-spinal fluid. The patient died a few hours after his admission to hospital. The right middle cerebral artery was intensely atheromatous and from its origin for the distance of one inch was dilated. Just beyond its commencement arose a large globular aneurysm with a fibrous wall containing old and recent laminated clot (Figure VIII). The sac was situated under the inferior surface of the right frontal lobe, the substance of which showed extensive necrosis and hæmorrhage. The aneurysm, or wall of the artery, had ruptured and the subdural and subarachnoid spaces and ventricles contained clot and blood-stained cerebro-spinal fluid. Both middle cerebral arteries were thickened and atheromatous, the heart was enlarged and the aorta showed a moderate degree of atheroma. Both kidneys presented evidence of old chronic inflammatory change.

Dr. Wright-Smith has recently given me the specimen from a wharf-labourer of seventy-four years, who collapsed at work and died almost immediately.

The patient had not seen a doctor for twenty years. There was extensive subarachnoid hæmorrhage, most evident at the base of the brain. The superficial cerebral vessels showed atherosclerosis. The right anterior cerebral artery proximal to the aneurysm was much larger than the left and very atheromatous (Figure IX). The middle cerebral arteries were very large and atheromatous; the anterior cerebral vessels distal to the aneurysm were small. It would appear as though the anterior wall of the anterior communicating artery, in the direct line of thrust of the blood stream in the large anterior cerebral vessel, had become dilated into an aneurysm, 1·0 centimetre (two-fifths

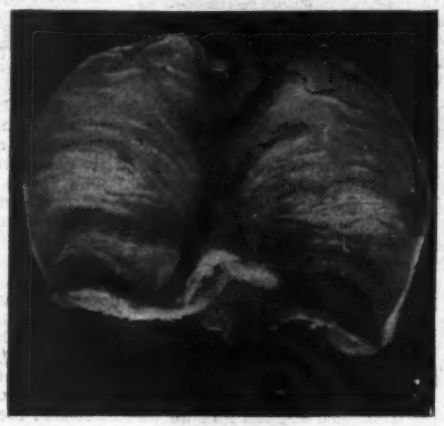


FIGURE VIII.

Arteriosclerotic aneurysm growing from an atheromatous and dilated middle cerebral artery. The sac is almost filled with laminated clot.

FIGURE IX.

Arteriosclerotic aneurysm growing from the anterior communicating artery. The gross atheroma of the basilar, internal carotid, middle cerebrals and anterior cerebral proximal to the aneurysm is well seen.

of an inch) in length, projecting between the anterior cerebral arteries. The fact that the vessels leading away from this area were small would subject this region to a very great stress. The sac of the aneurysm showed

intense atheroma, probably more marked than would occur if the aneurysm were a congenital one. Rupture had occurred at the heavily atheromatous apex of the aneurysm.

The heart was hypertrophied and the coronary arteries were atherosclerotic. The aorta showed gross atherosclerosis with ulceration and calcification. The renal, splenic and mesenteric arteries showed gross atherosclerosis with calcification. The kidneys appeared normal, except for the presence of a few small cysts.

W.D., a youth of 22 years, gave a life-long history of "bilious attacks", frequently dependent upon eating rich foods, cream, chocolate or pork. On the morning after a dietary indiscretion he would awake with pain over one or other eye, less frequently over both. The vision of the affected eye was frequently blurred and sometimes he would see coloured flashes of light. Spontaneous or induced vomiting relieved both headache and ocular manifestations.

In February, 1932, after eating much cream, he awoke with headache and vomited. This, however, gave but temporary relief, for later he was unable to read, on account of blurred vision. Towards evening he felt queer, remembers a "kind of whirl" and nothing more until he awoke next morning "paralysed" down the right side. On inquiry he stated that he was able to move the arm, but it seemed useless and he was unable to feel what was in his hand. He had difficulty in recalling and repeating words. In the evening he felt an indescribable epigastric sensation, which commenced slowly and then began rapidly to ascend until it reached his head, when he remembers thinking "I'm gone" and then lost consciousness. When he recovered, he felt numbness and weakness down the left side, which, like that on the right side, rapidly disappeared.

A month later he again lost consciousness, and when he came round, his left arm and leg were paralysed and he experienced a "frightful burning feeling" in the arm. An incomplete history led to a right fronto-parietal decompression being performed in March, 1932. No lesion was found, and subsequent treatment with deep X ray therapy was initiated.

He remained well until September, 1932, when persistent throbbing headache behind the right eye, nausea and repeated vomiting led to a generalized convulsion preceded by a queer feeling in the left arm and followed by transient weakness and profound disturbance of all forms of sensation over that arm. No further manifestations occurred. He commenced to suffer from two groups of symptoms early in 1935. There were attacks of pain, slight at first, but increasing to a very severe degree, referred to the feet and fingers, and followed by redness and extensive swelling, which lasted for six to eight days and then subsided, leaving a bluish, mottled, tender patch, which slowly disappeared (Figure X). Occasionally following severe pain a tense swelling would develop in the deeper tissues of the hand or leg. Independently of these there were attacks of numbness and loss of power in the left hand and left side of the face, lasting usually for a few days. Occasionally, for briefer periods, a similar disturbance would occur on the right side.

These two groups of symptoms were dissociated until in November, 1935, after a veritable shower of cutaneous lesions, he was troubled by frontal headache and epistaxis. Later, for a period of ten minutes, he felt "pins and needles" and slight numbness in the left thumb and index finger. In an hour's time this was repeated, to disappear and reappear regularly in a series of increasing attacks involving a progressively larger area—the hand and forearm, left side of the lips and left side of the tongue. The next day the headache was still present and the numbness gradually spread in synchronized attacks in the left arm and face. In between the attacks he could use his hand relatively well, but while the numbness was present he "kind of lost consciousness of anything in his hand"; it seemed paralysed and useless. The periods of numbness became of greater duration, and three days after their onset the attacks fused, so that dysæsthesia and disability were continuous. When anything touched his hand he noticed a burning feeling, which the next day extended over the forearm, and the day following over the whole arm. Speech became slurred and the left corner of his mouth dropped.

He was readmitted to hospital on December 1, 1935. Examination of the cranial nerves revealed a subjective alteration of sensation around the left corner of the mouth. He missed some heavy and some light stimuli over this region. Pin-point was felt equally sharply on both sides, but felt "different", as did heat and cold. The ability to distinguish between single and double simultaneous contacts was absent over the left side of the lips, normal over the right side. There was slight left-sided facial weakness and the tongue was protruded slightly to the left of the mid-line.

The motor power was good, but he was slower in initiating contraction on the left side. When the eyes were closed the outstretched left arm gradually fell, and there were piano-playing movements of the fingers. The muscles of the left arm were hypotonic.

The tendon reflexes in the left arm were diminished. Knee and ankle jerks were equal and active, the abdominal reflexes were normal and the plantar responses flexor. On sensory examination it was found that light touch, pin-point and heat and cold were equally well appreciated, but felt "different" over the left hand. There was a gross defect in the interpretation of position and passive movement of the fingers of the left hand and a slighter defect

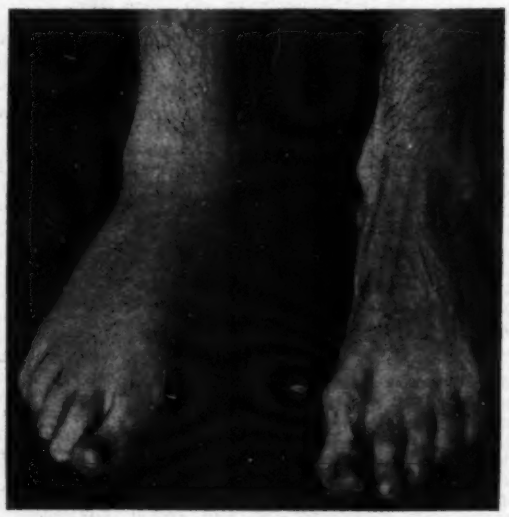


FIGURE X.

Photograph showing the swelling and obliteration of anatomical details due to lodging of an infective embolus in vessels of the right foot of W.D.

at the left wrist joint. He was unable to discriminate between two points and no threshold could be found. The apex beat was palpable in the fifth intercostal space, 8·75 centimetres (three and a half inches) from the mid-line. A soft systolic murmur was audible at all areas, but apparently it was less marked than at previous examinations. Rate and rhythm were normal.

Lumbar puncture revealed a clear colourless fluid under an initial pressure of 190 millimetres of cerebro-spinal fluid. Examination of cells revealed ten lymphocytes, one polymorphonuclear and one large mononuclear cell per cubic millimetre. The total protein content was 0·055%. The globulin was not increased. The colloidal gold test curve was 0000133330. The Wassermann test gave no reaction with both blood and cerebro-spinal fluid. Twenty days later the fluid contained seven lymphocytes, the total protein content was 0·04%, and the colloidal gold test curve was 0000000000. On examination of a gastric

test meal no free hydrochloric acid was found. X ray examination of the skull revealed no abnormality, apart from the decompression aperture. The urine contained albumin. The blood urea was 24 milligrammes per 100 cubic centimetres. The erythrocytes numbered 4,690,000 per cubic millimetre; the hæmoglobin value was 54%; the colour index was 0·57. The leucocytes numbered 11,700 per cubic millimetre. The differential leucocyte count was as follows: polymorphonuclear neutrophile cells, 49·5%; eosinophile cells, 1·5%; basophile cells, 1%; old metamyelocytes, 18%; monocytes, 5%; small lymphocytes, 25%. Mr. R. Douglas Wright kindly made accurate measurements of the temperature of a limb at the height of an attack of redness and swelling with thermo-electric couples. He concluded that "the affected area showed vasodilatation, which was increased by peripheral nerve anæsthesia. The whole periphery of the limb presented vaso-dilatation with a greater rise in surface temperature than on the affected area, but there was less exudation from the vessels. It was therefore a simple inflammation with the associated 'flare'."

Attacks of redness and swelling continued over the feet and the patient once complained of pain over the left side of the lower part of the chest. On February 21, 1936, he complained that he did not know where his left arm was. He then suddenly put his hands to his head and shouted: "Oh, my head!" He said that he felt as though something had burst inside his head, and complained of violent right-sided parietal headache. He then lost consciousness and four generalized convulsions occurred. The decompression region bulged. Lumbar puncture revealed a fluid under pressure of 270 millimetres, containing 4,500 erythrcoytes, 143 polymorphonuclear cells, 15 lymphocytes and 13 large mononuclear cells per cubic millimetre. The disproportion between red and white cells immediately after the onset of a hæmorrhage is interesting. The temperature rose to 40° C. (104° F.). During the next forty-eight hours the patient showed remarkable improvement and there were no residual abnormal neurological signs.

On March 24, 1936, after a restless night he awoke feeling as if his head would burst and experienced severe pain down the back of his neck. Suddenly his left side became limp and he lost consciousness; only the right limbs were moved subsequently. Two hours after the onset the right pupil was dilated and fixed, the left-sided deep reflexes were more active than the right, the abdominal reflexes were absent, and the plantar responses extensor. There was marked stiffness of the neck. His temperature was 36·8° C. (98·2° F.), his pulse rate was 92 and his respiratory rate 18. His systolic blood pressure was 130 and his diastolic pressure 60 millimetres of mercury. Lumbar puncture revealed a pressure much in excess of 300 millimetres and a deeply blood-stained fluid. He became cyanosed; the left side remained flaccid, in spite of accesses of tone affecting the right side; the left pupil became dilated and fixed, and death quickly followed.

The history provides scope for much fascinating speculation. Although the diagnosis of migraine has frequently been made in patients suffering from intracranial aneurysm, there can be no doubt that in this patient the early attacks were true migraine. Then in 1932, following upon what the patient believed to be an ordinary attack of biliousness, he became unconscious and developed a transient lesion of the left post-central cortex, followed rapidly by a similar disturbance of the right side. Other attacks, with varied epileptic phenomena, auræ and sequelæ, usually referred to the left arm, occurred at intervals, until during 1935 he commenced to suffer from attacks of redness and swelling referred to the extremities. In spite of the historical relationship of the attacks to food, no evidence of definite hypersensitiveness was disclosed by protein tests, except for reactions to dust and feathers. The events were seen in their true relationship only when his sister stated that he had twice passed blood in his urine, on the first occasion before his operation.

Then with dramatic suddenness a subarachnoid hæmorrhage occurred, followed later by a second and fatal attack. Throughout his illness there had been no signs or symptoms of cardiac embarrassment. He had once while in hospital complained of pain over the left costal margin. Only after the hæmorrhage was his temperature elevated.

Post mortem examination, made by Dr. Wright-Smith, revealed organized adhesions between layers of pericardium over the left ventricle. The endocardium showed a little fibrosis and the mitral valves a little thickening. On the auricular surface there were three small red vegetations of bacterial endocarditis. The spleen showed one infarct and the right kidney three infarcts, which were drying in.

In the right lateral sulcus on the lateral surface of the brain, one inch behind the Sylvian point, on the right middle cerebral artery there was an aneurysm, 1·25 centimetres (half an inch) in diameter, firmly adherent to the *dura mater*, under the posterior edge of the decompression (Figure XI). It had ruptured into the temporal

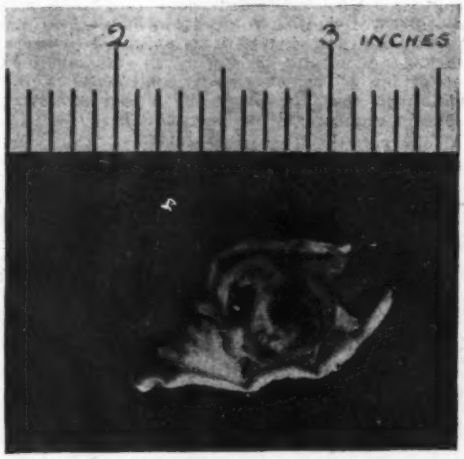


FIGURE XI.
Mycotic aneurysm growing from the middle cerebral artery and attached to the dura in the patient W.D.

lobe, which was greatly expanded, and its white matter was ploughed up by blood, which eventually reached the ventricle. Each ventricle contained blood clot accurately moulded to its contour. In the white matter of the left occipital region posterior to the posterior horn of the lateral ventricle there was a small area of degeneration about six millimetres (one-quarter of an inch) in diameter. Otherwise this hemisphere was normal macroscopically and microscopically.

Microscopically the myocardium showed patchy degeneration and fibrosis. There were well-marked fibrosis and vascularization of the cusps of the mitral valve. No Aschoff bodies were seen.

The wall of the aneurysm gave evidence of diffuse inflammation — fibroblasts were numerous, thin-walled new vessels could be seen, and the tissues appeared œdematous with patchy infiltration with lymphocytes and mononuclear cells. There were old and recent hæmorrhages in the surrounding tissue, scavenger cells filled with pigment being evident.

Fearnsides[8] states that at the London Hospital during the years 1907-1913, in a total of 7,924 *post mortem* examinations the head was opened in 5,432 cases. Fifty-one aneurysms of the cerebral arteries were found in 44 cases; 15 were due to infective embolism; 36 were congenital aneurysms. Death had been caused by rupture in 25 of the 31 patients with non-embolic aneurysm. Five patients died from other causes, and, as far as was known, the aneurysms had caused no definite manifestations during life.

In conclusion, one may refer to an article by one of the older teachers. Professor Brinton,[9] in 1851, found records in the literature of 40 cases of intracranial aneurysm, and he sums up the salient points as follows:

Age: On the average, forty-two.

Sex: Male to female as two to one.

Site: Anterior or carotid to posterior or vertebral system of vessels, equal; the basilar, one-third of the whole number.

Size: Half "filbert", one-fourth "pea", one-fourth "walnut".

Termination: In one-third only, rupture, or, more exactly, in three-eighths, rupture; in one-eighth, pressure, simple loss of functions; in one-eighth, congestion or hæmorrhage of brain; in one-eighth, inflammatory conditions of brain; in one-eighth, coincident disorders or accidents. In three instances the aneurysms were more than one in number; in one instance three were found.

Acknowledgements.

I wish to express my thanks to Dr. Charles Kellaway, Director of the Walter and Eliza Hall Institute of Research in Pathology and Medicine, for providing facilities for neuropathological study; to Dr. R. J. Wright-Smith, pathologist to the Royal Melbourne Hospital, for so generously making available the fresh material; and to my colleagues on the staff of the Royal Melbourne Hospital for allowing me to study patients under their care.

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OBSERVATIONS UPON THE METABOLISM OF CALCIUM AND PHOSPHORUS IN THREE CASES OF ACROMEGALY, ONE SHOWING OSTEOPOROSIS.

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In a survey of a small series of individuals suffering from acromegaly, undertaken in 1934, it was observed that the bony skeleton of one patient was unusually radio-translucent. All the bones, although conforming in size and contour to those found in acromegaly, displayed a marked degree of osteoporosis.

As no similar observation could be found in the literature, it was thought that this association was unique. Since that date Scriver and Bryan[1] have described an acromegalic patient showing a similar rarefaction of bone. So far as is known to us, no further observation of this sort has been published. Osteoporosis has, however, been described by Cushing[2] and others as a frequent association of the syndrome of the basophile adenoma of the pituitary gland. It was thought, therefore, of interest to examine the calcium and phosphorus metabolism of this patient and to contrast these results with those of other acromegalics, for the following reasons.

1. It is now well known that a certain number of patients suffering from parathyreoid adenomata show the marked rarefaction of bone characteristic of *osteitis fibrosa cystica*; moreover, in not a few cases removal of the tumour has greatly alleviated this condition. Such an occurrence has recently been reported in this country by Ross.[3] Furthermore, signs of hyperparathyreoidism have been found in association with parathyreoid enlargement, not definitely neoplastic.[4] This enlargement has been described as analogous to the hyperplasia of the thyreoid gland occurring in exophthalmic goitre, certain of these parathyreoid glands reaching a large size. Moreover, the histological appearances may differ from those observed in the ordinary adenoma; the cells are larger and the cytoplasm clear, so that the name *wasserhelle Zelle* has been appropriately applied.

2. Adenomata of various ductless glands have been observed in certain of the autopsies conducted upon acromegalics.

Although usually the parathyreoid glands have not been critically examined, large adenomata were observed in the case of Josefson[5] and in the recent case of Hadfield and Rogers.[6] In a survey of four cases of acromegaly that had come to autopsy Cushing and Davidoff[7] reported changes in the parathyreoid glands of two. In one, four para-

thyreoid glands were present, the largest measuring 1·0 by 0·8 by 0·5 centimetre. In the second the two upper glands were found to be slightly enlarged and each to contain a small central adenoma. In each case the thymus was enlarged and a large colloidal goitre was present. The changes in the parathyreoid glands cannot be said to have been conspicuous; nor were the skeletons observed to be rarefied. A rarefaction of the skeleton is more constantly found in association with the basophile adenoma of the pituitary gland. Too few autopsies have as yet been conducted on individuals harbouring this tumour to enable any general pronouncement to be made on the condition of the parathyreoid glands. They were enlarged in the case of Molineus,[8] one measuring 2·3 by 1·1 by 1·2 centimetres. In the recent well-studied case of Lawrence and Zimmerman,[9] a small adenoma was discovered. This patient was of interest in that the serum calcium was repeatedly found to be normal and the calcium balance was found to lie within normal limits; yet a marked general osteoporosis was present. The authors consider it reasonable to relate the adenoma of the parathyreoid to the skeletal decalcification, despite the normal biochemical findings.

The decalcification of bone associated with the basophile pituitary adenoma is often very striking. Thus a considerable loss of height may occur, and spontaneous fractures take place, the bones being so soft as to be easily cut with scissors at autopsy. This is unlike the rarefaction of bone occurring in *osteitis fibrosa cystica*, in that it is more general and is not accompanied by cyst formation or associated with tumours within the bone. Yet even with this pituitary adenoma it is difficult to be certain as to which internal secretion may be held responsible for any particular physical anomaly. Thus it is common to find at autopsy that more than one endocrine gland is hyperplastic or actually adenomatous.

Furthermore, a syndrome resembling that of the basophile adenoma may occur with adenomata of the adrenal cortex. The case described by Calder and Porro[10] is of great significance. Here, although osteoporosis was extreme and an adenoma of one adrenal cortex was discovered, serial section of the pituitary gland showed no increase of basophile cells, an observation confirmed by Cushing. A single normal parathyreoid gland was found.

The relation of decalcification of bone to parathyreoid hyperfunction is therefore not so simple as may at first appear.

Although it is usual to find a high serum calcium value in *osteitis fibrosa cystica*, there is still some doubt whether it invariably accompanies hyperparathyreoidism. A mild general osteoporotic form may occur, the bones being identical in appearance with those found in association with the basophile pituitary adenoma and other conditions.

Thus Albright, Aub and Bauer[11] reported seventeen cases of hyperparathyreoidism. Their Case II was of this mild type and not associated with any cystic changes in the bone. The patient, when first seen, showed normal serum calcium values. It was not until repeated determinations had been made over a period of one year that the correct diagnosis was made. At operation a parathyreoid tumour measuring 1·0 by 0·7 by 0·5 centimetre was removed, with great symptomatic improvement.

It is apparent, then, that a mild hyperparathyreoidism may not be revealed by the simpler blood analyses. It also seems probable that the determination of the calcium and phosphorus balance, as at present carried out, may not invariably reveal this condition in the milder cases. Any conclusions as to the absence of excessive secretion of the parathyreoid glands must be made with reservations, even in the presence of an apparently normal calcium balance.

It is also possible that certain parathyreoid glands may not produce over-secretion continuously, and the biochemical examination may be undertaken during a phase of normal activity. It is also possible that certain parathyreoid glands may habitually produce over-secretion in the presence of some stimulus and show little histological evidence of so doing. Reference has been made to the generalized enlargement of the parathyreoid glands, which may result in the clinical signs of hyperparathyreoidism.[12][13]

Experimental Methods.

Experiments were carried out by one of us (I.McP.) in collaboration with the staff of the diet kitchen of the Alfred Hospital.

Collection of Specimens.

Three-day periods were used, the fæces from each period being marked off by giving a 0·3 gramme (five grain) carmine capsule with breakfast on the first day of that period. The specimens as obtained were sent as quickly as possible to the laboratory, where they were transferred to a jar containing a little hydrochloric acid and kept refrigerated till the end of the period. The whole specimen for the period was then dried in an electric oven at 100° to 110° C., weighed, pulverized, passed through a 20-mesh sieve, and bottled for analysis. Constipation was present in all patients and was controlled by the regular administration of cascara in doses just sufficient to produce one formed stool a day.

Twenty-four hour specimens of urine were collected at the patient's bedside into a bottle containing two cubic centimetres of formalin. At the completion of the collection of the sample the volume was measured and one-quarter was kept for analysis. The three samples obtained during a period were thoroughly mixed before sampling.

Diet.

William D. and Anne F. were placed on normal diets, adequate in all respects. The diet of Emma B. contained a minimum amount of calcium and a

normal quantity of phosphorus, after the methods of the Massachusetts workers.[12] The patients were admitted to wards for several days before analyses were commenced. During this time the diet was adjusted so that the patient's weight remained constant and bowel action was normal. It was recognized that the splanchnomegaly of acromegaly necessitates large bulk and that the caloric intake must be kept fairly high to avoid loss of weight. Fluid intake was kept constant; the fluids were given as water or tea, distilled water being used throughout.

Acid-base values varied from five cubic centimetres of normal acid to ten cubic centimetres of normal base, as calculated from the analyses of Sherman and Gettler[14] and Sherman.[15] Although an alkaline diet is said to lead to decalcification, such variations probably would not be sufficient to cause any considerable change in calcium and phosphorus metabolism.

In the preparation of the diet each article was weighed to 0·1 gramme, then cooked by steam and served in the same dish. At the same time an amount equal to one-tenth of the diet was sampled for analysis and collected into a weighed jar, and at the end of the period the whole sample was dried and treated exactly as were the fæces.

Methods of Analysis.

The food, fæces and urine for each period were analysed for calcium, phosphorus and nitrogen.

The nitrogen was determined in food and fæces by a macro-Kjeldahl procedure. Urinary analyses were done either by the macro-Kjeldahl or the micro-method of Young.[16] Although a slight amount of nitrogen may be lost during the drying of food and fæces, the figures are sufficiently accurate for the detection of a definite loss or of a retention. In this connexion also a careful check was kept on the patient's weight.

Calcium and Phosphorus.—Food and fæces were ashed by the Stolte method (described by Peters and van Slyke[17]), 5 to 8 grammes of fæces and 20 to 25 grammes of food being used. The ash was dissolved in 10 cubic centimetres of 10% hydrochloric acid and filtered into 100 cubic centimetre flasks, the crucible then being washed repeatedly with a further 15 cubic centimetres of 10% hydrochloric acid, followed by hot water, and the washings were poured through the filter. Calcium and phosphorus were determined in aliquot portions of this solution.

All phosphorus determinations were done by the method of Mathison,[18] modified by Mackay and Butler.[19] The calcium in the food and fæces of the patients receiving adequate calcium was estimated by the method of Washburn and Shear,[20] and that in their urine by the method of Shohl and of Pedley.[21] When the diet was poor in calcium the calcium in both excreta and food was estimated in specimens containing from one to two milligrammes by a modification of the micro-method described by Hansman and Wilson;[22] the oxalate was precipitated by the addition of one cubic centimetre of 5 N ammonium chloride and one cubic centimetre of 10% oxalic acid, and the final volume made up to ten cubic centimetres. For each analysis of urine twenty cubic centimetres were first ashed with nitric acid and the residue was extracted with six cubic centimetres of 2·5% hydrochloric acid.

Blood Analysis.—Clark and Collip's modification[23] of the Kramer-Tisdall method was used for serum calcium. Inorganic phosphorus was determined by the method of Bell and Doisy, as modified by Benedict and Theis.[24]

Case I.

Emma B., a nurse, was first seen by one of us (L.B.C.) on February 15, 1929. At this time her age was forty-eight years.

She had suffered from a poisoned finger twenty years previously and attributed much of her subsequent ill-health to this. She had not felt well since. A photograph taken at that time showed her to be a healthy-looking young woman of good appearance, her features showing no trace of acromegaly. After this illness she always felt tired, had little energy and did little nursing for some years.

While nursing about twelve years previously she developed an acute mental breakdown and was confined to a home for two years. At about this period her appearance was noticed to have altered.

FIGURE I.
Patient I, showing the acromegalic features and the pitting of the skin.

Menstruation had ceased at the age of thirty-five. She had not suffered from hypersomnia, but had frequent bouts of insomnia. She was sensitive and retiring by disposition at this period, spending most of her time by herself. Only occasionally did she suffer from headaches.

On examination she presented the appearances of advanced acromegaly (Figure I). Her nose was long and

broad, her lips were coarse and prominent, her tongue was large. The skin of the face was deeply pitted. The hands were not only broader and coarser than those of the examiner, but the fingers were at least one inch longer. The finger nails were twice the width of those of the examiner. The feet were not so grossly enlarged in comparison with the hands.

A gross kyphosis of the spine was present. The ribs exhibited beading at their junction with the cartilages. No abnormality of thoracic or abdominal viscera was noted. The palpable arteries were not thickened; the systolic blood pressure was 140 millimetres of mercury. The urine contained neither albumin nor sugar.

A full neurological examination revealed no abnormality. The vision was $^6/_6$ in each eye. The optic disks were normal. The visual fields were full.

An X ray film showed the pituitary fossa to be considerably enlarged. Within the *sella turcica* scattered areas of calcification could be observed. The frontal sinuses and mastoid air cells showed an unusual degree of pneumatization. Although not especially noted at the time, a review of this older film showed the bones of the calvarium to have been rarefied.

Since then she has been under occasional observation, her appearance remaining substantially the same. A recent X ray examination has revealed no further enlargement of the pituitary fossa. The visual fields remain normal. She has developed no signs of *diabetes insipidus* and no hypersomnia. On occasions she has had headaches. She has suffered at times from flatulence and has had periods of diarrhœa. Of late years she has complained of pains in the back and about the hips.

In 1934, in a review of her condition it was noticed that the bones of the skull were definitely rarefied (Figure II). The calvarium showed a uniform fine granular mottling, the appearance suggesting that of the surface of a cut pear. The cortices of the bones were much narrowed. Other bones when examined showed a uniform rarefaction (Figure III). Thus the phalanges of the hands, besides showing the usual arrow-head appearances of the distal portions, were definitely rarefied. The epiphyseal portions were trabeculated, the cortices of the shafts being much narrowed. The carpus was rarefied, as were the bones of the feet. Osteophytes were present in association with the bones of the metacarpus, the metatarsus and the phalanges. Such long bones as were examined presented similar changes. The head of the femur, besides exhibiting many osteophytes, was rarefied. The bones of the pelvis and such portions of the spine as were included in the films showed substantially the same changes.

At this time the thyreoid gland and the neighbouring structures were carefully examined, but were not found to present any detectable abnormality.

It was decided to investigate, as far as was possible, the metabolism of calcium and phosphorus in this patient. It was realized that in all probability the eosinophile adenoma was not progressing. Thus it was known that no clinical progression had occurred for at least six years. Moreover, the calcification visible within the pituitary fossa also suggested some regression in the tumour.

The patient was placed on a 1,566 calorie diet, adequate in all respects except calcium, the intake of which was 0·17 gramme per day. In the first three periods the average calcium intake was 0·51 gramme and the average output 0·93 gramme, showing a negative balance of 0·40 gramme and a total calcium excretion of 0·0129 gramme per kilogram per period. The figures thus fell within the normal limits given by Bauer, Albright and Aub[128] for this type of diet. Calcium intake was then increased to about the amount which had been found to be excreted. After a lapse of three days a further period of analysis showed the patient to be in equilibrium with regard to calcium. Phosphorus was in balance during periods I to III, but became slightly negative in period V. The paths of excretion of these elements were normal, 62% to 69% of the total calcium output and 22% to 32% of total phosphorus output being in the fæces.

The serum calcium was 10·0 milligrammes *per centum* on two occasions and the inorganic phosphorus 5·0 milligrammes *per centum*. (See Table I.)

TABLE I.

Subject.	Weight in Kilograms.	Period.	Nitrogen, in Grammes, per Three-Day Period.					Phosphorus, in Grammes, per Three-Day Period.						Calcium, in Grammes, per Three-Day Period.						Serum Calcium.
			Intake.	Urine.	Fæces.	Total Output.	Balance.	Intake.	Urine.	Fæces.	Total Output.	Balance.	Balance Corrected for Nitrogen Loss.	Percentage Output in Fæces.	Intake.	Urine.	Fæces.	Total Output.	Balance.	Percentage Output in Fæces.	Excretion per Kilogram.	
Emma B. ..	72·0	1	32·0	30·7	4 4	35·1	−3·1	2·30	1·54	0·72	2·26	+0·04		32	0·49	0·34	0·57	0·91	−0·42	63	0·0126	10·0
	72·0	2	35·1	32·6		37·1	−2·0	2·23	1·57		2·12	+0·11		26	0·55	0·30		0·95	−0·40	68	0·0132	
	72·0	3	32·4	30·5	4·5[1]	35·0	−2·6	2·32	1·85	0·55[1]	2·40	−0·08		23	0·49	0·29	0·65[1]	0·94	−0·45	69	0·0131	
Average ..	72·0		33·2	31·3	4·5	35·8	−2·6	2·28	1·65	0·61	2·26	+0·02		27	0·51	0·31	0·62	0·93	−0·42	67	0·0129	
	72·0	4																				
	72·0	5	36·7	35·6	3·2	38·8	−2·1	3·56	2·21	0·62	2·83	−0·27		22	0·93	0·37	0·60	0·97	−0·04	62	0·0135	10·0
Anne P. ..	67·7	1	38·9	38·9	2·8	41·7	−2·8	3·75	2·89	0·16	3·05	+0·10		21	2·22	0·85	0·72	1·57	+0·65	46	0·0233	10·3
	67·3	2	42·1	38·1	2·8	40·9	+1·2	3·66	2·97	0·78	3·75	+0·11		21	2·34	1·01	0·76	1·77	+0·58	47	0·0263	
	67·3	3	41·3	36·5	4·7	41·2	+0·1	3·77	2·67	0·99	3·66	+0·11		27	2·25	0·90	1·42	2·32	−0·07	61	0·0345	
Average ..	67·4		40·8	37·8	3·1	40·9	−0·1	3·79	2·84	0·84	3·68	+0·11		23	2·27	0·92	0·97	1·89	+0·38	50	0·0260	
William D. ..	99·5	1	40·8	45·2	4·6	50·0	−9·2	3·37	3·35	1·00	4·35	−0·98	−0·45	23	2·02	1·23	1·17	2·39	−0·37	49	0·0240	10·9
	98·6	2	38·1	44·6	5·3	50·1	−12·0	3·32	3·81	1·03	4·84	−1·52	−0·83	25	2·99	1·225	1·94	3·16	−0·87	61	0·0320	
	97·7	3	40·8	45·1	5·6	50·7	−9·9	3·97	3·40	1·13	4·53	−0·56	0	25	2·74	1·25	1·52	2·77	−0·03	55	0·0283	
Average ..	98·6		39·9	45·0	5·2	50·2	−10·3	3·55	3·52	1·05	4·57	−1·02	−0·43	24	2·35	1·23	1·54	2·77	−0·42	55	0·0281	

[1] Owing to the difficulty of separating the stools of periods 2 and 3, they were analysed together and the results averaged.

The basal metabolic rate and glucose tolerance curves were normal. Renal tests showed a slightly lowered efficiency, with the urea clearance 60% of normal, blood urea 31 milligrammes *per centum*, and urea concentration test 1·7% in the first hour (65 cubic centimetres passed) and 1·95% in the second hour (48 cubic centimetres passed).

These data, therefore, offer no evidence of any alteration in the metabolism of calcium or of phosphorus. The excretion of calcium per kilogram per period averaged 0·0129 gramme, as compared with an average figure of 0·0120 gramme obtained by Bauer, Albright and Aub on thirteen normal individuals on a slightly lower calcium intake than this case. Their results varied from 0·007 to 0·016 gramme per kilogram per period.

Case II.

Anne F., a married woman, aged thirty-nine years, was first seen by one of us (L.B.C.) on August 29, 1934. A general alteration in her features and a broadening of the hands and feet had been noticed for an indefinite period. She did not suffer from headaches, but had suffered from pains in the back and in the legs for some months. She had been married for nine years, but had no children. Menstruation had ceased four years previously. She had experienced no polyuria or excessive thirst. She was not excessively sleepy, although seven years previously for some time she had felt a strong desire to sleep after her mid-day meal. At this time she considered that she had been unnaturally drowsy. Her general health was good and she was able to follow fairly heavy work in her household duties as a farmer's wife. She had had no serious ill-health in the past.

On examination she was of a typical acromegalic appearance (Figure IV). Her face and forehead were broad. The mandibles, although not grossly enlarged, showed a broad spur-like ridge on each lateral surface, near the angles. The nose was broad. The skin of the face was smooth and soft, except over the nose, where it was slightly pitted. The tongue was large, broad and fissured at the edges. The hands were short and broad and the fingers markedly broadened. The feet were short, but broadened in the distal half.

No abnormality of any organ could be determined. The systolic blood pressure was 170 and the diastolic pressure 115 millimetres of mercury. The heart was rapid, possibly owing to her apprehensive state. A full neurological examination revealed no abnormality. The optic disks were normal in appearance, the fields were full, and vision was $^6/_6$ in each eye. The pituitary fossa was slightly enlarged, one side of the floor being depressed into the sphenoidal sinus. The bony skeleton showed no osteoporosis.

She was given a course of deep X ray therapy shortly after she was first examined.

The patient was placed on a diet adequate in all respects, with a daily calcium intake of 0·76 gramme. Definite retention of calcium occurred in periods I and II, the amount retained being correlated with low fæcal values (47% of the total output). The urinary values remained fairly constant, so that this patient appeared to be able to store a considerable amount of calcium. This was possibly due to a previous calcium depletion, as the patient stated that she had never taken milk in any form. The effects of such a low intake might be exaggerated by the acromegalic overgrowth. That such a compensatory effect occurs after calcium starvation is suggested by the experiments of Albright, Bauer, Roper and Aub[20] on parathormone. In period III calcium was in balance. Much looser, bulkier stools eventuated, indicating rapid passage through the alimentary tract. These contained more nitrogen, calcium and phosphorus, while urinary nitrogen and phosphorus were correspondingly decreased. This illustrates the importance of maintaining normal stools. The average calcium excretion per kilogram per period was 0·028 gramme, with an average retention of 0·38 gramme. Phosphorus was in equilibrium throughout and showed an average excretion per kilogram per period of 0·0546 gramme. (See Table I.) The serum calcium was 10·3 milligramme *per centum*. Glucose tolerance was slightly lowered. Fasting blood sugar was 0·11%; blood sugar half an hour after 50 grammes of glucose was 0·13%; one hour after glucose it was 0·13%; and two hours after glucose it was 0·14%. No glycosuria was found.

The excretion of calcium was within normal limits. Sherman,[19] in an individual receiving 0·39 gramme of calcium daily, found an average excretion of 1·59 gramme per period, equivalent to 0·026 gramme per kilogram. He also states that the optimum requirements per day per 70 kilograms weight are 0·68 gramme of calcium and 1·32 grammes of phosphorus. With the subject in balance these intakes would result in the excretion per kilogram per three-day period of 0·029 gramme of calcium and 0·0566 gramme of phosphorus.

FIGURE IV.
Patient II, showing the acromegalic features and broadening of the hands.

Case III.

William D., a married man, aged forty years, was first seen by one of us (L.B.C.) on November 29, 1933. For twelve years he had noticed a progressive enlargement of his hands, feet and head, so that he periodically required larger boots and hats. He had suffered from no real headaches, but remarked that at times at night, when he turned over in bed, he felt as though there were sand in his head. He was sexually potent and had four children; the youngest was aged three years. He had noticed that his left testicle was small and that the right had increased in size. He had no polyuria or increased thirst. He did not suffer from undue drowsiness at that time, although he complained of this later in 1935, when it was on occasions so pronounced that his tools of trade might drop from his hand when he was at work. His general health was good in 1933. In 1935 he complained of breathlessness and of lassitude. In this year also he noticed some failure in the sexual function.

On examination he presented all the appearances of advanced acromegaly (Figure V). His lower jaw was enormously enlarged. The skin of the face and head was

thick and deeply pitted and lay over the scalp in numerous coarse folds. The tongue was grossly hypertrophied. The hands and feet were markedly deformed, the fingers and toes being much broadened.

No abnormality of heart or lungs was noted. The urine contained neither albumin nor sugar. The abdominal viscera showed no enlargement on palpation. The optic disks were normal in appearance. The visual fields were full. Vision was $^6/_6$ in each eye. The skiagram revealed a large enclosed pituitary fossa of about the size of a large cherry.

A course of deep X ray therapy was given in 1934.

He was placed on a 2,425 calorie diet, adequate in all respects and containing 0·77 gramme of calcium a day, but owing to the difficulty in obtaining his cooperation the caloric intake was insufficient to prevent loss of weight. It was necessary, therefore, to make a correction for the phosphorus lost in the liberation of protein (N : P = 17·4).

FIGURE V.
Patient III, showing the advanced acromegalic features and great enlargement of the hands.

In period I there was a loss of all elements, which became more marked in period II. Here an unexplained rise in the excretion of phosphorus in the urine and of calcium in the fæces was associated with an increased loss of nitrogen. In period III, however, calcium and phosphorus were in equilibrium. The patient was therefore in balance on an intake per period of 2·74 grammes of calcium and 3·97 grammes of phosphorus, or 0·0283 gramme of calcium and 0·0462 gramme of phosphorus per kilogram per period. Such amounts are not excessive and we regard the calcium metabolism of this patient as essentially normal. The fæcal excretion of calcium varied from 49% to 61%, and of phosphorus from 23% to 25% of the total output. The serum contained 10·9 milligrammes of calcium and 5·0 milligrames of inorganic phosphorus *per centum*. (See Table I.)

Discussion.

In the three cases of acromegaly here described the modern methods of analysis did not reveal any disturbance in the metabolism of calcium or of phosphorus. Nor were these elements present in the serum in concentrations differing from those accepted as normal.

Of these three cases, Case I may be described as advanced and almost certainly non-progressive, showing a marked general osteoporosis and resembling the type encountered in certain cases of hyperparathyreoidism. Case II is early and probably progressive, showing no osteoporosis. Case III is advanced and progressive, also showing no osteoporosis.

In comparing the two advanced cases, I and III, both show the usual bony changes of their disease, in that the bones have increased in size, have widened by the laying down of new bone beneath the periosteum, and many new bony formations are present. In Case I, however, the bones, having attained the usual contour of those found in acromegaly, yet have undergone rarefaction. Thus the calvarium, which is much increased in width, is definitely rarefied and has not the density usual in acromegaly. The thickened cortices of the long bones have undergone a similar rarefaction, a process which has even extended to the osteophytes.

To what may this calcium loss be due? An immediate difference is to be observed between these two cases; thus it is the progressive case which shows the usual increase in density of bone, while the non-progressive case shows rarefaction. The factor of progression, or continued activity of the acidophile adenoma, cannot be lightly dismissed when considering this difference. It is possible that, had the secretion which is responsible for the new production of bone been continuously present in the blood of Case I, rarefaction might not have occurred. As far, however, as is known to us, a comparable loss of density of bone has not been noted in those acromegalics who have undergone successful operation and in whom the pituitary secretion may be presumed to have been reduced. Moreover, the translucency of the bones in Case I is much greater than is observed in bones of even average width in normal individuals. Hence a simple cessation of the secretion of the acidophile cells of the adenoma cannot be accepted as a complete explanation of the osteoporosis.

It may be suggested that a deficiency of calcium in the diet may have resulted in a call being made upon the bony tissues. This patient, as far as could be determined, did not habitually take a diet deficient in calcium. It is also to be noted that in Case II the patient, whose diet did not contain any notable quantity of milk, did not show these changes. This case, however, was probably progressive, a factor to be considered, as has been observed.

Scriver and Bryan[1] accept a dietetic factor as the most likely explanation of the osteoporosis occurring in their acromegalic patient. They believe that the call for the new production of bone was not met by an adequate supply of calcium; hence

the bone was rarefied. It would seem to us that exception may be taken to this view. Should it be a prominent factor, it would be likely that many cases of acromegaly would show a similar rarefaction. As is well known, this does not occur.

A generalized decalcification of bone is known to occur in thyreotoxicosis. In this condition the excretion of calcium is raised. No evidence of thyreotoxicosis was observed in the case described.

In the light of such knowledge as we possess as to the causes of generalized osteoporosis, it would seem likely that the decalcification in Case I has resulted from some imbalance of the endocrine glands. It is admitted that the biochemical investigations do not support this view. In support of this suggestion, one may again refer to the case of basophile adenoma reported by Lawrence and Zimmerman.[9] Here a small parathyreoid adenoma was present in association with osteoporosis; yet the serum calcium was normal and the calcium balance was within normal limits. In view of the association of the parathyreoid secretion with the mobilization of calcium, the possibility must be considered that in Case I the parathyreoid glands were over-active either intermittently or continuously, and that this aberrant function could not be detected by the biochemical methods used. Furthermore, it is possible that whilst hyperfunction of the parathyreoids may have been present during the progressive phase of the pituitary tumour, a normal state may have existed during the retrogression of the latter. Moreover, it cannot be dogmatically stated that the secretions of pituitary and parathyreoid glands are alone necessary for the mobilization of calcium. It is to be remembered that in the case of Calder and Porro[10] an adenoma of the adrenal gland alone was discovered.

Finally, it must be remembered that our knowledge of the exact chronic state in which calcium exists in the blood and tissue fluids is far from complete. It is possible that studies in variations of the amount of calcium that is physiologically active may throw some light on the problem that we have just discussed.

Summary.

1. The metabolism of calcium and phosphorus was investigated in three acromegalic persons. In none was any abnormality detected.

2. A considerable rarefaction of bone was observed in one case, judged to be non-progressive.

3. The possible relation of bone rarefaction to endocrine and other changes is briefly discussed.

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THE INDUCTOTHERM.

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Physics.

The inductotherm is a vacuum tube oscillator which generates a high-frequency current of about twelve megacycles per second. If the current from the inductotherm is passed through a coil, an alternating magnetic field of the same frequency will be set up. If then any conductor is placed within this magnetic field, voltages will be induced in that conductor, as a result of which induced currents will flow (these are known as eddy currents) and will produce heat in the conductor. This is known as eddy current heating or heating by magnetic induction.

If the conductor is any part of the human body it will of course be heated, the heat produced being in direct proportion to the intensity of the current and the number of turns of the coil; but heat will not be produced at the same rate in all tissues; the greatest heat will be produced in the tissues with the greatest conductivity. This fact gives the inductotherm its special value as a heating agent. The most conductive tissues in the body are the most vascular tissues, and the blood stream itself; skin, fat and bone are poor conductors, and so are not nearly so much heated when subjected to the same intensity of current.

Compare this with the other two methods of electrical heating: (*a*) diathermy, and (*b*) short and ultra-short wave apparatus.

In diathermy the heating produced by any given current is roughly in accordance with Joule's law, that is, in proportion to the resistance of the conductor. When applied to the human body, this means that the skin and superficial fat are unduly heated, while the deeper vascular tissues may not be heated as much as may be desired. In actual practice this was a definite limitation of the benefits of diathermy. Of course in spite of this handicap diathermy was of very great benefit, but must now be considered a poor second best when it is desired to heat the more conductive tissues.

The short wave apparatus heats tissue placed in the high-frequency condenser field between the electrodes of a short wave generator. The formula to determine the maximum rate of heating in tissues of varying resistance, when placed in a condenser field of a given frequency, is complicated. The practical results, stated simply, are that in a condenser field of 25,000,000 cycles (twelve metres wave-length) per second, there will be a maximum heating of tissues with the same resistance as that of a 0·05% saline solution.

As the frequency of the condenser field rises, maximum heating takes place in tissues with less resistance, that is, with more conductivity. But for the maximum heating to occur in tissues with the same resistance as in normal saline solution (that is, the body fluids) the frequency would have to be 350,000,000 cycles per second (0·85 metre wave-length).

In the present state of electrical knowledge it is impossible to make machines to produce a current of such a frequency with enough energy output to be of any value in the treatment of human beings. So that at the present moment it would seem that the inductotherm gives us the best form of heat for the treatment of disease. Some workers with short wave apparatus (high frequency condenser field) claim a special destructive action on bacteria at varying frequencies; they claim that such action is due to some specific electrical effect on the bacteria. This, if it is confirmed, is of course of immense importance; but the great majority of authorities believe that the beneficial effects of the various forms of electrical heating are due to heat alone, and not to any mysterious electrical action on molecules and atoms. In any case at present the only demonstrable effect of the passage of the currents through the body is the production of heat.

Technique.

The use of the inductotherm is simplicity itself. The current is led by a heavily insulated flexible cable from the machine to the part to be treated, and then back to the machine. There are several ways of arranging the cable for treatment. If a limb or a joint is to be treated, from two to four turns of the cable are passed round it; if a portion of the trunk is to be treated the cable is wound into a "pancake coil", that is three or four turns of the cable in a flattened spiral are placed against the part, the size of the "pancake" depending, of course, on the size of the field to be heated.

An alternative way of heating a limb is to place it between the sides of a long U of cable, the turn of the U crossing the limb. I found a useful method of heating all the limbs and of raising the body temperature, which I have not seen described. The patient sits facing the machine; the cable is led down to the feet, wound two or three times round them, and led back to the machine. The patient lightly holds the cable leading from machine to feet with one hand, and the cable leading from feet back to machine with the other.

Treatment can be given without the removal of ordinary clothing, but any metal article in the field of treatment must be removed, as it will become very hot. Additional insulation, generally four to eight thicknesses of a towel or sheet between the skin and cables, is desirable. No large mass of metal should be near the coil during treatment, as a great deal of power would be lost.

The use of the inductotherm is by no means fool-proof; superficial burns can easily be caused, unless reasonable care is taken, and the patient should always be asked to report at once any feeling of uncomfortable heat.

It has been noted that the greatest heat is generated in the more conductive, that is the more vascular tissues, and the least in skin, fat and bone. That is strictly true if all the conditions are equal. One of the most important of these conditions is distance from the source of energy, that is, the coil carrying the current. Obviously the skin and subcutaneous fat must always be closer to the coil than the deeply seated tissues. In actual practice the skin will always receive more heat than any muscle or internal organ, and provided there is no disturbance

of sensation, the sensory nerves will always give warning of too much heat.

Besides the switch for turning the current on and off, the machine has only one control for increasing and decreasing the current; on this is marked an arbitrary scale from one to sixteen. In the average case the cable is arranged as desired, and the control is turned to sixteen; in about three minutes the control is turned down to thirteen, and left at that setting for the rest of the treatment.

The duration of a treatment is about half an hour, which I have found to be equal to an hour's diathermy.

Watson & Sons imported my machine for me at the end of 1934. I have been using it constantly since then, and my considered opinion is that it is a great deal more efficient than diathermy in every case in which heat is beneficial. In many conditions in which it is not desirable to disturb the patient it is enormously superior. For instance in pneumonia the whole of the lungs can be treated without even disturbing the bed-clothes; that is an advantage quite apart from its greater efficiency in heat production.

Medical diathermy, with the one exception of auto-condensation for high blood pressure, has served its very useful turn, and should step aside from its place in treatment to its place in history.

In hospital practice the inductotherm should very greatly increase the efficiency of treatment, as hospitals do so grudge time. I have had a number of patients from metropolitan hospitals who have had long courses of treatment with diathermy, half an hour three times a week, when they should have been having three-quarters of an hour six times a week, and the results have invariably been poor. When will hospital authorities learn that to be miserly with time in electrotherapy is a waste, not a saving? Surely it is better to treat fifty patients efficiently than a hundred inefficiently.

The inductotherm may also be used for the production of artificial fever for the treatment of general paralysis of the insane, and for any other condition in which a considerable rise in body temperature might be useful.

The technique is quite simple; the patient is wrapped in two or three blankets, the cable is passed three times loosely round the waist, and the patient is then wrapped in extra blankets and mackintosh sheets. Care must be taken that the cable does not press closely against the body, or burns may be caused. Pressure is most easily avoided by using a divided mattress and separating the two parts at the waist. The patient's temperature can be kept at 40·5° or 41·1° C. (105° or 106° F.) for from five to seven hours, or whatever time may be desired.

An alternative method is to place a large "pancake" coil both back and front of the patient; the two "pancakes" must be wound in the same direction, that is, both clockwise, or both anti-clockwise.

Experiments.

I carried out a simple if rough-and-ready experiment to compare the heating efficiency of diathermy and the inductotherm. The experiments were carried out under exactly similar conditions.

1. The bladder was emptied into a glass, and the temperature of the urine was found to be 35·6° C. (96° F.).

2. A coil of the inductotherm was placed above the pubes, and the current was turned on; as much heat as was comfortable was kept on for thirty minutes, and the bladder then emptied. The temperature of the urine was 40·2° C. (104·5° F.).

3. Two lead plates 20·0 by 12·5 centimetres (eight by five inches) were placed one above the pubes and one behind the sacrum, and connected to the diathermy machine, which was turned on to give as much heat as was comfortable for thirty minutes. The temperature of the urine when passed was just over 35·6° C. (96° F.).

No claim of scientific accuracy is made for the experiment, but it does show that the inductotherm can considerably heat an internal organ containing a conducting fluid.

Cases.

CASE I.—I recently had a severe teno-synovitis of the right wrist joint, affecting chiefly the tendons of the extensors of the wrist and the *supinator longus*.

As it began as a moderate ache the day after I had done a lot of painting, I thought it was merely a "mild strain", following an unaccustomed use of muscles. During the next two days it got steadily worse, and movements of extension and supination were really painful, and at that stage creaking, when the affected tendons moved, first became obvious. The creak could easily be felt and heard by another person, and the dorsal aspect of the joint was a good deal swollen.

I continued to do my ordinary work and to drive a motor car, of course sparing my right wrist as much as possible. Neither splint nor bandage was used, the sole form of treatment being half an hour's application of the inductotherm once a day. The relief following each treatment was very noticeable, movement of the joint being much less painful for a considerable time. Definite improvement began about the third day of treatment, and after the fifth day treatment was stopped and there was no further trouble.

Probably if it had been practicable to give the tendons rest with the usual splint as well, healing would have been more rapid still.

CASE II.—An old gentleman, of about seventy-five, had had a chronic arthritis of both shoulder joints for many years; there was a fairly constant ache, and the range of movement of the joints was limited by pain.

He had previously had thorough courses of treatment by both diathermy and infra-red radiation, with only slight temporary relief.

He had daily treatment for three weeks with the inductotherm, the usual half-hour exposure being given each time. After the first week he began to get relief from the ache, and the range of movement increased. At the end of the course he had very little aching and almost full movement, without pain. That was over a year ago, and his condition has not relapsed.

CASE III.—A young woman with chronic pyelitis and cystitis had had every kind of treatment, except removal of the infected kidney, during the past twelve years. She had frequent severe attacks of lumbar and suprapubic pain. Electro-therapy was tried in the hope of relieving pain, not with any expectation of curing the condition. Diathermy was used repeatedly and gave no relief whatever. During an acute flare-up of the pyelitis, when she was confined to bed with fever, and had much pain, repeated exposures to infra-red rays were given, as it was easy to take the machine to the patient. She got much relief from it, but on all occasions when the attack was not severe enough to confine her to bed the inductotherm was used, and nearly always gave ease from pain for several hours after an exposure. By having frequent treatments she was able to avoid a great deal of misery which she would otherwise have suffered.

Infra-red radiation, and still more the inductotherm, were the only things that gave her ease, except constant doses of powerful hypnotics; and heat was certainly preferable to drugs, besides acting more quickly and efficiently.

Many other cases could be quoted, but I do not wish to prolong the article unnecessarily.

After a year and a half of constant use of the inductotherm I am convinced that in practice, as in theory, it is a much more efficient producer of heat in deeply-seated tissues than diathermy.

Reports of Cases.

FRACTURE OF THE STERNUM.

By ERIC H. GOULSTON, M.B., B.S., F.R.C.S.I., F.R.A.C.S.,
Sydney.

CASES of fracture of the sternum are rare. The following is published to show that union with some deformity has caused no inconvenience and has led to quite a fair result.

W.A., aged forty-eight years, was gaily riding his bicycle on December 5, 1935, when a stick lodged between the spokes of the front wheel, precipitating him forward onto the ground. His head came into violent contact with the ground and his body was suddenly bent forward in acute flexion. He suffered great pain in breathing or moving and kept his body bent forwards. There was considerable localized tenderness over the front of the chest wall, with deformity at the manubriosternal junction; his respirations were shallow and rapid. He was conveyed home and an X ray examination was made on the following day; he was still in severe pain, aggravated now by a cough. There was no lesion of the skull or spine. He was placed on fracture boards with a firm pillow between the shoulders, and reduction was attempted by traction and counter-traction through hyperextension of the body. His dyspnœa was greatly relieved, and in the absence of complications his condition gradually improved, and on January 22, 1936, he was able to walk about freely without a support. On March 23 he resumed work and recommenced cycling.

The X ray report was that no fracture of the ribs was seen. The sternum was fractured in two places; one fracture was situated just below the *manubrium sterni* and the other midway along the body of the sternum. The upper fragment was displaced inwards near the manubrium, where there was also a completely separated fragment; this was displaced forwards.

Acknowledgement.

I am indebted to Dr. S. Elphinstone for the X ray films.

Reviews.

LAWRENCE'S "DIABETIC LIFE".

MANY practitioners can recall the pleasurable satisfaction we experienced when Dr. Lawrence's book, "The Diabetic Life", first appeared in 1925. Written at a time when insulin was new and rather feared, it at once assumed its present position of being the most lucid, compact and practical exposition of the fundamentals of diabetic knowledge in existence.

Though intended essentially for the general practitioner, its latest appearance covers the subject so adequately that even the general physician requires to seek no further for current diabetic information.[1] The most outstanding commentary, both on the excellence of the book and the rapid growth of diabetic knowledge, is that between 1925 and 1936 it has reached its ninth edition and has been reprinted in no less than four foreign languages. Nevertheless, Dr. Lawrence has not found it necessary to make any great change in the original arrangement nor to depart from his ingenious "line-ration" scheme, except that, in accordance with modern ideas, his black lines now each contain ten grammes of carbohydrate instead of five. In his hands, and with most intelligent patients, this method has proved successful. Medical practitioners who have to deal with large numbers of public hospital patients generally find that the prescription of "individual" diets in their final amounts and arrangement leads to less misunderstanding. Once such a patient knows his drill, he can be taught food equivalents and alternatives. Dr. Lawrence's diets are less generous in carbohydrate than those advocated by Rabinowitch or Sansum, but the line-ration scheme can be readily applied to give similar low fat/high carbohydrate ratios. The present edition is rich in illuminating diagrams illustrative of the twenty-four hour blood sugar curve in treated and untreated patients. Patients receiving insulin are also charted similarly, and these charts show the real necessity for such an examination before a diabetic can be considered to be adequately under control. Lists of new food values determined by Dr. McCance show a change, particularly as regards British fruits.

The treatment of minor complications and interruptions to the diabetic life is most important from the general practitioner's viewpoint and receives special mention. Some additions have been made to the list of recipes, which appear most appetising. Valuable paragraphs, such as "The Omission of Insulin", "Insulin-Resistant Cases", "Difficulties in Injection", show anticipation by the writer of nearly every practical difficulty encountered by either patient or physician. The book is becoming perhaps a little too wide for the average diabetic to encompass, but its well-known lucidity and logical arrangement will continue to retain the lay reader's interest. A final paragraph on protamine insulinate ends with the following wise warning: "These new developments will neither cure diabetes nor remove the present necessity of injections in severe diabetes."

FUNGI AS PATHOGENIC AGENTS.

APART from infection by ringworm and the saprophytic or subparasitic growth of various fungi on the tongue, elsewhere in the oral cavity, in the external auditory meatus, and on moist locations, as between the toes, invasion of the tissues of man by fungi higher than the bacteria is not common. Nevertheless such invasion may lead to extensive and even fatal lesions and may require much skill and knowledge to enable a correct diagnosis to be made and so the appropriate treatment to be applied. We have seen nodules of *Aspergillus* from the pleural cavity, a large mass of compacted fungal hyphæ loose in a cavity in the lung, hyphæ in sections of lymph glands, and cases of fatal invasion of the retroperitoneal tissues and of the brain and meninges by *Cryptococcus* (*Torula*). "Medical Mycology",[1] by Professor C. W. Dodge, of Washington University, is therefore a work to be welcomed by all those on whom may fall the task of recognizing and of identifying such fungal agents of disease. The medical microbiologist, with his attention focused more on the relationships to disease than on the systematic position in the animal or plant kingdom of the parasitic agents concerned, cannot be expected to have more than a passing knowledge of the latter aspect. In the work under review, Professor Dodge has collected, in a large volume of 900 pages, all references available to him of instances of disease in man due to invasion by fungi, and, as far as possible, has assigned these fungi to their appropriate positions in the scheme of systematic mycology. Full descriptions are given of the microscopic features of these species, with cultural characteristics when available, and keys to enable them to be more readily identified.

[1] "The Diabetic Life: Its Control by Diet and Insulin: A Concise Practical Manual for Practitioners and Patients", by R. D. Lawrence, M.A., M.D., F.R.C.P.; Ninth Edition; 1936. London: J. and A. Churchill. Demy 8vo, pp. 232. Price: 8s. 6d. net.

[1] "Medical Mycology: Fungous Diseases of Men and Other Mammals", by Carroll William Dodge, Ph.D.; 1935. St. Louis: The C. V. Mosby Company; Melbourne: W. Ramsay (Surgical) Proprietary Limited. Super royal 8vo, pp. 900, with illustrations. Price: £3 3s.

The work opens with a chapter on the general morphology of the fungi, followed by one dealing with their physiology; and then come chapters on the preparation of culture media, the methods of isolation, mounting media, staining methods, and so on. In Chapter VI are given the international rules of botanical nomenclature. Then follows a systematic description of the various pathogenic fungi, with references to their associations with human disease. An extensive bibliography is given at the end of the chapters, in which we see appropriate references to various recorded Australian cases.

This work is a very valuable one for the laboratory worker who may be called upon to identify some fungoid growth associated with diseased conditions. There has been much difficulty in identifying specifically many of these organisms, but Professor Dodge's work will simplify matters considerably by bringing together the descriptions of the various species of pathogenic importance. This work is therefore a welcome addition to the literature on the subject.

MENTAL NURSING.

THE second edition of "Mental Nursing (Simplified)" is hardly justified by the claim of its author (Dr. O. P. Napier Pearn) that there are 150 reasons why it "should be more useful than the first edition". "One hundred additional facts" and "twenty-five minor alterations and improvements" totalled, with "25 subjects . . . freshly introduced or given more attention", to the mathematical accuracy of 150, scarcely make one sufficient reason for recommending to mental nurses the use of the new edition. The style and method do not appeal, and the same fantastic representations fail to convey the serious meanings which they are intended to imply. It is unlikely that this book will either oust or assist in the understanding of the "Handbook for Mental Nurses".

ANATOMY AND ITS STUDY.

THOSE who are interested in the framing of a medical curriculum will appreciate Professor S. E. Whitnall's small volume, "The Study of Anatomy".[2]

The work covers only a little over 100 pages, divided into seven chapters.

In the first chapter, on the nature and principles of the subject, Professor Whitnall explains for the benefit of the student the various subdivisions included in the subject of anatomy and their interrelationship. Emphasis is laid on the fact that students of medicine are biologists, with man as their subject, and that human anatomy is a study not of the dead, but of the living.

In the second chapter there is sound advice as to the methods to be pursued during the actual course of dissection; this may be summed up in the author's own words: "Know the body rather than the book."

Chapter III concerns text-books and how to use them, while Chapters VI and VII also deal with books. Chapter VI deals with books for general reading. The author quotes the well-known Osler's "Bedside Library" and wonders how many of these books would really hold the student's mind. Agreement or disagreement here will probably depend upon the reader's particular make-up.

Chapter VII consists of an excellent bibliography.

Chapters IV and V, on teachers and lecturers and examinations respectively, should be read by teacher and student alike. Though short, they are replete with common-sense ideas. As a sample, Arnold, of Rugby, is quoted as saying to his boys: "You have come here not to read, but to learn how to read."

[1] "Mental Nursing (Simplified)", by O. P. Napier Pearn, M.R.C.S., L.R.C.P., D.P.M.; Second Edition; 1936. London: Baillière, Tindall and Cox. Crown 8vo, pp. 312, with illustrations. Price: 5s. net.

[2] "The Study of Anatomy: Written for the Medical Student", by S. E. Whitnall, M.A., M.D., B.Ch., M.R.C.S., L.R.C.P., F.R.S.; Third Edition, revised and enlarged; 1936. London: Edward Arnold and Company. Crown 8vo, pp. 113. Price: 4s. 6d. net.

Discussing this work as a whole, we conclude that the author has met most of the criticisms of the teaching of anatomy in recent years. In developing his thesis he has made reference to the writings of many who have advanced helpful criticism in this respect. The book should be read by all those about to undertake the study of anatomy. If they take heed of the author's advice they will find their study pleasant and interesting. The aim of the author is to insure that the student should obtain a thorough knowledge of the anatomy of the living. If this goal is achieved, many of the pitfalls of practice will be avoided. The author, throughout the book, exhibits a true love of his subject, and for those who read it aright there will be the inspiration that may lead to great things.

A HANDBOOK OF UROLOGY.

THE subject of urology has been admirably epitomized in "A Handbook of Urology for Students and Practitioners", by Vernon Pennell.[1] As the author points out, diseases of certain systems tend to gravitate to special hospitals, and so the student is in danger of missing preliminary training in special work, and too often this work is regarded as essentially post-graduate in nature. General practitioners are not expected to deal with the many urological problems that demand special training and equipment, but they must appreciate the importance of symptomless hæmaturia, they must be able to deal in an adequate, albeit temporary, manner with such problems as acute retention of urine, and to treat the routine complaints of general practice, such as prostatitis, cystitis and stricture. The hand-book would appear to take the student and practitioner rather further than is necessary in some aspects of the subject. Ureteral transplantation is considered in more detail than most other procedures, as also is the operation of Harris for prostatectomy, and although admittedly the latter is accorded the position it deserves, the everyday problem of chronic retention is more important and in a book of this type should have been considered at greater length.

The book is well set out and abounds with practical points in technique that so often bridge the gap between success and failure in a surgical manœuvre.

Some faults are apparent. "Abrodil" only is mentioned as a medium in excretion urography. The statement is hardly correct that retrograde pyelography has been largely superseded by the excretion method. In most cases the older method must still be regarded as the ultimate means of diagnosis. The few diagrams in connexion with pyelography are poor. The usefulness of the indigo-carmine test in estimating general functional capacity of the kidneys is not mentioned. The cystoscopic lithotrite only is mentioned. However, most of the subject matter is excellent, and we feel that the author has achieved his objective of placing before students and practitioners a concise, logical and readable aid in the urological problems with which they are most often confronted.

Notes on Books, Current Journals and New Appliances.

A DICTIONARY FOR NURSES.

THE fourth edition of "A New Dictionary for Nurses", by Sister Lois Oakes, has been published.[2] This is a useful little book that may well be given to nursing trainees. In addition to the 346 pages devoted to the dictionary proper, there are several appendices on diet, urine testing, poisons and so on, that make the book more valuable.

[1] "A Handbook of Urology for Students and Practitioners", by V. Pennell, M.A., M.B., B.Chir., F.R.C.S.; 1936. Cambridge: The University Press; Melbourne: S. Jaboor. Crown 8vo, pp. 232, with illustrations. Price: 7s. 6d. net.

[2] "A New Dictionary for Nurses", by L. Oakes, S.R.N., D.N.; Fourth Edition; 1936. Edinburgh: E. and S. Livingstone. Demy 16mo, pp. 388, with illustrations. Price: 3s. net.

The Medical Journal of Australia

SATURDAY, SEPTEMBER 19, 1936.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

THE MEETING OF THE FEDERAL COUNCIL.

In the report of the Federal Council of the British Medical Association in Australia, published in this issue, reference is made to matters of considerable moment. Some of the problems are those of the Branches as part of one Australian body; others concern the profession in its pursuit of scientific progress; others again have to do with the profession and its relation to the community. The Federal Council is the central executive body of the Australian Branches; each and every member should thus be conversant with its decisions, with the reasons for making those decisions, and with their significance. Hence it is perhaps well that attention should specially be drawn to some of the more important matters discussed at the recent meeting. At the same time the report published in this journal does not give an adequate idea of the thought and energy that are given to problems as they arise.

The appointment of a subcommittee to report on the cost of a federal secretariat will be welcomed. The officials of the Parent Association, when they were in Australia about a year ago, recognized the need for the strengthening of the bonds between the Australian Branches, and this no doubt influenced the Home Council in making its generous grant to the Federal Council. Members of the Australian Branches must not close their eyes to the possible needs of the future. That the members of the Federal Council are alive to some of the difficulties that Australian practitioners may have to face in the years to come is shown by the repeated reference to the possible introduction of national insurance and to the need for studying it. National insurance is bound to engage the attention of the Federal Council before very long, and any secretary who was appointed by the Council would have to give much attention to the subject. In any matter such as this the medical profession in Australia will have to be able to speak with one voice and its members will have to know beforehand what they want to say. A permanent secretariat cannot be run and important issues such as national insurance cannot be met without the expenditure of money. The Parent Association has given a grant of one thousand pounds a year for three years, but the Branches must recognize that more money than this will be needed and that they will have to find it.

The discussion about the proposed National Health and Medical Research Council showed that the Federal Council had not departed from the views held by the Federal Committee ten years ago. It was wise enough, however, to agree to cooperate with the new body. On a previous occasion we have pointed out that the National Health and Medical Research Council will bring together Commonwealth and States in one common endeavour—the pursuit of medical research. The official recognition of the need for research in medicine is most encouraging; it shows that the Federal Government is not concerned only with the present, but with the future health and welfare of the people of the Commonwealth. It would have been foolish, or more than foolish, if the Federal Council had refused to cooperate in the scheme because its own ideas had not been adopted in their entirety at the outset. In most large undertakings started on apparently well-ordered lines adjustments and

changes are made as they are indicated by working conditions. The proposed new body will deal with public health matters as well as with research. We cannot imagine that anyone appointed to this council would be deliberately obstructive, nor is it likely that those members who were not thoroughly conversant with medical research would refuse to accept the advice of those who were. As far as research is concerned, we have no doubt that the council will be guided by those who know most about it, possibly by an executive of research workers, as the Federal Council has suggested.

That further progress has not been made with the framing of regulations for the formation of special sections within the Association is to be regretted. Twelve months have elapsed since the subject was discussed with the officials of the Parent Body and finality has not been reached. Next August the fifth session of the Australasian Medical Congress (British Medical Association) will be held, and it seems likely that at least one group of practitioners will at that time wish to form a special section. These sections, it will be remembered, are to include members of all six Branches, and membership will be by election. In other words, only recognized followers of a specialty will be eligible for membership of a section devoted to the study of that specialty. We would urge the Federal Council at its next meeting to draw up regulations and to take any other steps that may be necessary to enable those who wish to form special sections to take action next August.

Among the other important subjects discussed at the meeting of the Federal Council were medical registration and reciprocity between the public hospitals of the several States. Reciprocity between hospitals should be attained without much difficulty; the uniform drafting of medical acts of the States will be more difficult.

Current Comment.

POST-OPERATIVE RESPIRATORY COMPLICATIONS.

PULMONARY complications following surgical operations are always important and sometimes dangerous, and from time to time surveys of the subject appear in medical literature. The figures presented in such reports are not always helpful, as they may be modified by so great a variety of factors, but nevertheless there is some value in carefully collected statistics. E. A. Rovenstine and J. B. Taylor have collected the figures for the Wisconsin General Hospital for the years 1933-1934, using an improved anæsthetic record.[1] They exclude from consideration minor procedures carried out with local infiltration anæsthesia only, and also operations performed solely under chloroform or barbituric acid derivatives, since the number of these was small. The anæsthetics used in this series of 7,874 operations were ether, nitrous oxide, ethylene, tribromethanol, "Procaine" and "Cyclopropane". Premedication was usually given in the form of morphine or scopolamine, and in a few cases barbiturates. All the patients were seen prior to operation by a member of the department of anæsthesia and were revisited on the third to the fifth day after operation, and subsequently if complications were present; no definite time limit was observed. The more important complications were as follows: pneumonia, lobar or bronchial, 49 cases (0·6%); massive pulmonary collapse, 13 (0·2%); partial collapse, 22 (0·3%); bronchitis, 22 (0·3%); and laryngitis, 144 (1·8%). In addition, over 360 patients suffered from some grade of cough, apparently without actual pulmonary signs being present.

It was found that during the months in which respiratory diseases were more prevalent a greater number of complications occurred. This would be expected, as would also the finding that the chance of a patient suffering respiratory complications after operation was almost doubled by the previous existence of a pharyngitis, either acute or chronic, or of oral sepsis; the existence of a cough before operation raised the number of complicated cases by 25%. The influence of the anæsthetic agent used did not seem to be of great importance. It is recognized, of course, that certain anæsthetics are safer in emergency operations on enfeebled subjects, so that statistics are invalidated in this respect; but when the types of cases were subdivided according to the expected degree of operative risk, it was found that the various anæsthetics were mutually comparable as regards morbidity rates. It was found, of course, that nitrous oxide and "Procaine" presented better figures for the bad risk groups, but it is interesting and also cheering to the man in average practice to learn that the general figures for ether are good, and that in the emergency cases the figures for ether can compare with any other agent except the spinal and infiltration techniques when "Procaine" is used.

The degree of narcosis in relation to respiratory complications is difficult to assess, especially with such widely differing anæsthetics. But the authors found that a distinct difference could be detected in the morbidity rates when deeper grades of narcosis

[1] *The American Journal of the Medical Sciences*, June, 1936.

were employed; the more profound the narcosis, the greater the degree of post-operative risk of respiratory troubles. Still more evident was the influence of the duration of operation, which seems to be of greater importance than the intrinsic gravity of the operation. When the operation lasted over an hour the respiratory complications were doubled in the succeeding half-hour, and actually trebled in the second hour; while, when the procedure lasted three hours, more than 31% of the patients suffered some respiratory complication.

The authors remark in conclusion that their statistics show well the influence upon post-operative morbidity exerted by the skill and experience of the anæsthetist. Despite all the recent advances in anæsthesia, this last factor is worth stressing. Safety as regards the avoidance of post-operative pneumonia, for example, does not rest only with the sturdiness of the patient or the ability of the surgeon or the appropriateness of the particular anæsthetic chosen; it also depends upon the personal skill of the anæsthetist. Adequate training of students in anæsthesia should be recognized as a very important part of any scheme of medical training, either before or after graduation.

THE TREATMENT OF HIGH BLOOD PRESSURE.

PERHAPS so much has been written about "blood pressure", as the radio pundits call it, that everyone is weary of the topic, save those who ride some new hobby of drugs or diet. But the addiction of the public to mysterious remedies is deep-rooted and is reflected even in the willingness of a certain section of the medical profession to try the latest in tablets or ampoules. John Hay, whose knowledge of cardio-vascular disease secures him a ready hearing, has written a simple and well-balanced article on "The Treatment of Patients with Abnormal Blood Pressure".[1] The title of this paper should be noted; he does not set out to treat a "disease" by more or less specific routine. Indeed he admits, as all must, that a specific therapy or even an effective therapy for hyperpiesia is unknown. Yet he would not have us adopt a helpless attitude, for the man rather than the malady may be handled so as to increase his safety and prolong his life, while his more distressing symptoms may be diminished, even if not entirely controlled.

Hay points out that the first thing to do is to make a searching inquiry into the patient's life in order to discover any adverse factors in his past, his present life or his habits. Next he lays down several simple rules. When high blood pressure has existed for some time it is likely that the constant pressure observed represents the optimum for this individual patient's organs, and therefore should not be lowered drastically. Next he points out that the well-being of the patient depends upon his maintenance of a vigorous heart muscle; this is likely to be adversely affected by several factors, the most important of these being obesity, anæmia and the presence of any toxic state. As a matter of fact, experienced clinicians will admit that it is in the assessing of these factors that judgement is so necessary and, further, that it is not always easy to arrive at a balanced opinion. Hay advises small, dry meals, which should include meat in moderation and fresh vegetables and fruit. When the patient actually complains of symptoms, such as those of an early cardiac failure, or vertigo, or paræsthesiæ *et cetera*, a period of rest is desirable with some sedative drugs. At this stage it may be necessary to "edit" the patient's duties, and Hay makes the practical suggestion that the physician should obtain from the patient a list of everything that makes a call on his time and energies, applying the blue pencil to each one that is not necessary for his living or his pleasure in life. Obesity should be treated *secundum artem*; this few will overlook, but infection is a more subtle thing and is worth a few words. Hay regards every toxic illness as serious, even influenza or tonsillitis, and advises that these small indispositions should be treated with the respect that is their due and that what he has said concerning the importance of maintaining a capable myocardium should not be forgotten. In convalescence hæmatinics may be of special value to counter the slight anæmia which may then be seen if watched for carefully. Other general suggestions that he makes are the use of potassium iodide, sanctioned by several generations of physicians, the combination of bromide and valerian for the highly strung, particularly the woman at the menopause, and the occasional value of a weekly dose of a mild mercurial purge. Venesection, though only of transient effect as regards the actual level of the blood pressure, has its place also in the severer grades of hyperpiesia, to relieve vertigo, tinnitus or mental confusion, and also the more serious pulmonary œdema that may occur in the course of the all-too-common cardiac failure. Lastly, the possibility of a hyperthyreoidism in the hyperpietic patient should be borne in mind.

It will be said that there is nothing new in all this; perhaps that is its value. Let it be noted that Hay has said nothing of vaso-dilators, and that the following of his advice will not enrich the manufacturing chemists. Let us remember that our medical fathers doubtless recognized and treated hyperpiesia with reasonable success, even though they knew nothing of sphygmomanometers. It is also certain that their treatment would include not only all that is wisest in the dietetic restrictions that are advised today, but also the basic methods of the up-to-date psychological expert, and that the few drugs they used were practically identical with those used by the authority we cite here. Though it may be depressing, we may close with Hay's quotation from Major: "If our knowledge of ætiology of arterial hypertension is shrouded in a certain haze, our knowledge of an effective therapy in this disease is enveloped in a thick fog."

[1] *The Practitioner*, June, 1936.

Abstracts from Current Medical Literature.

MEDICINE.

Thyreoid Gland and Atherosclerosis.

A CERTAIN amount of experimental work has been done on the relation of atheromatosis and the function of the thyreoid gland. Niels P. Dungal (*The Lancet*, June 13, 1936) records two observations that he has made while working as a pathologist in Iceland. First, he has noted the weight of the thyreoid gland as seen *post mortem* in 57 individuals over the age of twenty years, the average for both sexes being 13·3 grammes. Comparison with figures of other countries shows that this is very low. Japan is the next on the list, with an average weight of 16·4 grammes. This weight has been explained by the small stature of the people of Japan; but that argument does not hold for the Icelanders, who are among the tallest people in Europe. There are no signs of dysthyreosis, which would be expected if the thyreoid gland was actually too small. It is suggested that the high iodine intake by the Icelander is correlated with the size of the gland; fish forms a large portion of the diet, and the soil is alkaline and of volcanic origin, the iodine content being therefore high. The second observation is the rarity of atherosclerosis seen at autopsy in comparison with what is seen under similar conditions in other countries. Detailed comparison on this point is difficult, but the fact is important. In view of the proved experimental relation between the thyreoid gland and atheromatosis, it is suggested that the abundance of iodine in the food, soil and air is responsible for both the observed facts.

Leuco-Erythroblastosis.

J. MCMICHAEL AND J. W. MCNEE (*Edinburgh Medical Journal*, May, 1936) report the clinical and pathological condition of three patients presenting as examples of splenomegaly of unknown origin and described under the term leuco-erythroblastosis. The constant presence in the circulating blood of numbers of immature cells, both red and white, is characteristic of a number of well-recognized blood diseases, such as myeloid leuchæmia; anæmia is associated with the latter condition, not necessarily with the condition under discussion. A brief history of the dyscrasia is given. Vaughan has shown the close association of leuco-erythroblastic anæmia with diseases affecting the bones, such as myelomatosis, secondary carcinomatosis, osteosclerosis, congenital or acquired, and the curious porosity of bones in children described by Cooley. Five case reports from the literature are given in which there is no obvious cause for the condition. The authors detail three cases of their own and suggest that the condition should be recognized definitely by ordinary clinico-pathological methods from other forms of splenomegaly. All three patients were women; they are all dead; one died from aspiration pneumonia following splenectomy; the other two cases terminated with the features of the hæmorrhagic diathesis. The presenting symptom in each instance was splenomegaly, which is due to myeloid metaplasia. The disease appears to be uniformly fatal and its course is uninfluenced by X rays, liver therapy and splenectomy. Experimentally and clinically, myeloid metaplasia occurs in conditions in which there is an enormous call on the hæmopoietic reserves of the body, with which the marrow is unable to cope. It would be expected, then, that myeloid metaplasia would occur either as a synergic effort along with an intensely active bone marrow or else as a compensatory effort when there was no possibility of further marrow extension. Why such extensive extramedullary hæmopoiesis should be found in the presence of a bone marrow which in places was almost aplastic, it is impossible to determine. Unfortunately, only one of the three cases here reported came to autopsy, and there the femur contained no obvious red marrow; microscopically there were small accumulations of erythroblastic and leucoblastic cells.

Acid Ammonium Phosphate as Urinary Acidifier.

STANLEY ALSTEAD (*Edinburgh Medical Journal*, May, 1936) reports the results of his investigations into the difficulties of acidifying the urine, the necessity for which arises from the use of hexamine and ketogenic acids in the treatment of urinary infections. The buffer action of the sodium phosphates prevents any considerable fluctuation in the hydrogen-ion concentration. Acid sodium phosphate has been in common use as a urinary acidifier since its recommendation by Hutchison, but later work has shown that its effect is insignificant and unreliable. Acid ammonium phosphate, on the other hand, tends to make the urine more acid and the pH more regular. Ammonium chloride and calcium chloride have been used with success, but the gastric irritation following their prolonged administration is a disadvantage. The author carried out observations on thirty-four consecutive patients; a normal diet was given, but the daily calorific intake and proportion of protein, carbohydrate and fat remained fixed for each individual throughout the experiment. The effect of administration of acid sodium phosphate and acid ammonium phosphate was noted in each case by varying the doses given and by determining the pH and titratable acidity. The sodium salt was uncertain in its action and even alkalinized the urine in some cases; its use should therefore be abandoned. The ammonium salt in doses of 2·0 grammes (thirty grains) thrice daily diminished the pH and no further increase in the hydrogen ion concentration occurred with doses greater than this. The maximum acidity in the majority of patients was represented by a pH of 5·5. Considerable alteration was frequently seen, a difference of 3·0 in the pH being noted on one occasion. Very little change follows in an already acid urine; it is pointed out that this is an academic point only. The cathartic action of the ammonium salt is not obvious when the minimum doses necessary for the efficient acidification of the urine are administered.

Diabetes and Insulin.

GUSTAV SINGER (*Münchener medizinische Wochenschrift*, May 1, 1936) expounds his view that insulin treatment of diabetes is by no means an unmixed blessing. He claims that an insulin habit, insulinismus, is established, with lowering of the glucose tolerance through suppression of the endogenous production of insulin. This produces ill-effects on the heart and circulation, and also subjects the patient to the dangers of hypoglycæmia. In the last ten years, that is, the insulin era, the mortality from diabetes has risen enormously, and especially in the United States of America. The increase is manifest after middle age, with a marked preponderance in females after the age of thirty-five years. And there has been a greatly increased mortality from diabetic coma, not only in Germany, but also in the United States of Amercia. Many of these patients with coma, having been treated with insulin for several years, had become less responsive to even large doses of insulin. A rational and economical treatment of diabetes connotes emancipation from general insulin treatment, which can no longer be based on the theory that the pancreas is the main factor in the pathogenesis of diabetes. It is known that other glands (pituitary, thyreoid) are often concerned. Diabetes may be fatal with a healthy pancreas. A pancreas reduced to one-tenth of its substance can supply the insulin requirements of the body. In hepatic diabetes the pancreas may be intact. There are many insulin-resistant cases in which the specific (insulin) treatment is inapplicable. Juvenile diabetes is probably purely pancreatic diabetes, and in this form of the disease insulin is a blessing and a necessity. And for the relief of coma and of the severest stages of diabetes, insulin, used for a short period, certainly does wonders. More than ten years ago the author elaborated a treatment by parenteral injections of non-irritating (that is, non-fever-provoking) proteins, beginning with very small doses and gradually increasing. By the end of six weeks there is a substantial improvement of the metabolism, as evidenced by

the diminution or disappearance of the glycosuria and the reduction of the blood sugar, often to normal. If the result is not satisfactory, a second course is given; and if vegetable protein is used in the first course, an animal protein in the second will have a more active effect. In the majority of cases, originally severe, such an improvement occurs after two courses of this treatment that the enhanced tolerance permits an increase of carbohydrate to approximately the amount in a normal diet. This improvement persists for months and years. The author's recommendations may be summarized as follows: (i) Simplify and standardize (1,700 to 1,900 calories) the diet. (ii) Ration insulin in courses of short duration, except in juvenile cases. (iii) Investigate possible pathological conditions of the central nervous system, and endocrine or metabolic disturbances (liver *et cetera*). (iv) Use non-specific protein injections. For further details of his protein injections the author refers to several earlier publications of his own, which are listed at the end of the article.

The Termination of Artificial Pneumothorax.

JOHN CHICHESTER DUNDEE (*The British Journal of Tuberculosis*, April, 1936) discusses the correct time to cease giving refills in the artificial pneumothorax treatment of pulmonary tuberculosis. He demands a minimum period of three years after the last positive sputum, or after the final radiological evidence of the disappearance of a cavity. A very slow and gradual reexpansion is desirable. Thus if in a lesion of the upper third of the lung an 80% collapse is deemed necessary, by the end of the second year reexpansion may find a collapse of 50%. By the end of the third year there may be only a 10% or 20% collapse, and at this time special care is necessary. Serial skiagrams taken during the whole period of treatment should now be examined. Usually the most recent film will reveal calcified deposits and striations and bands in the site of the original lesion. If the serial films do not reveal resolution or calcification or fibrosis, the probability is great that reexpansion will result in reactivity. The younger the patient, the longer should the treatment be maintained; and the author has not yet allowed reexpansion in the treatment of a patient in his teens. The patient who has to return to manual labour should have pneumothorax treatment considerably longer than the sedentary worker, and in some instances the treatment should be continued indefinitely. So also the poor patient is likely to be less able to develop resistance by virtue of insanitary surroundings, inadequate food and lack of fresh air. In conclusion, the author insists that the final sputum test before refills cease should be by guinea-pig inoculation. Two months before the last refill is due a three-day specimen of sputum is inoculated into a guinea-pig. Of examinations which otherwise reveal no abnormality, 10% are by this test proved to be positive. Though it is costly, the author considers that this is justified as a safeguard both to patients and their contacts.

Mercurin.

M. N. FULTON (*The New England Journal of Medicine*, May 28, 1936) discusses the treatment of œdema by mercurin suppositories. Mercurin or mercupurin is the sodium salt of trimethylcyclopentane dicarboxylic acid-methoxy-mercuryhydroxide allylamide. It is administered in the form of suppositories in the œdema of cardiac failure. One gramme of ammonium chloride is given three or four times a day prior to and during treatment. Five hundred milligrammes of mercurin are given as a suppository in the morning after a cleansing enema every four to six days. Twenty-five patients have been treated. Diuresis was observed in all but six of these patients, the amount of urine passed being similar to the amount passed after intravenous or intramuscular injection of "Salyrgan". Mercurin is ineffective in the ascites of cirrhosis of the liver. No untoward effects were observed.

Protamine Insulin.

E. P. JOSLIN *et alii* (*The New England Journal of Medicine*, May 28, 1936) record some results of treatment of diabetic patients with protamine insulin. Protamine insulin is prepared by injecting one cubic centimetre of protamine derived from fish sperm into a standard solution of insulin; a cloudy solution results, which is injected with or in place of insulin before breakfast or the evening meal. In new patients protamine insulin alone was injected, the number of units employed being similar to the usual dose of old insulin. Protamine insulin acts slowly; its full effect may not be manifest for five or six days. At first glycosuria increased, but after five or six days the glucose disappeared. One hundred to two hundred and fifty grammes of carbohydrate were given. Hypoglycæmia may occur after protamine insulin from eight to twenty-four hours after the injection; the hypoglycæmic symptoms come on more slowly, but there may be a series of attacks. The effect of protamine insulin lasts at least twenty-four hours, so that a cumulative effect may be observed. There is some evidence that protamine insulin deteriorates after four or five days.

Coma in Malaria.

A. SLATINEANO *et alii* (*Archives roumaines de pathologie expérimentale et de microbiologie*, June, 1936) publish a histopathological study of malarial coma in a mother and fœtus aged four months. The mother, aged twenty-five years, died. The *Plasmodium falciparum* and *Plasmodium malariæ* were found in the blood before death. The outstanding changes were that the structure of the liver was entirely effaced, there were acute glomerulonephritis, pericarditis and hæmorrhages into the myocardium. All tissues contained numerous parasites in the vessels. The liver of the fœtus was affected similarly to that of the mother. The mother's brain showed thick meninges invaded by numerous mononuclear cells; the vessels were congested; the parasites were observed in the vessels. The brain showed intense glial proliferation, infiltration with mononuclear cells and muffing of the vessels with mononuclear cells (perivascular infiltration). The nerve ganglia and sciatic and crural nerves were invaded by pseudo-gummatous nodules and perivascular infiltration was marked. Pigment deposits were observed in all the organs studied.

Blood Platelets.

HÉLÈNE-G. IOAN (*Archives roumaines de pathologie expérimentale et de microbiologie*, June, 1936) describes studies on the variation in the numbers of platelets in infectious diseases. In most infectious diseases fever is accompanied by diminution in platelets, most marked when the infection is severe; from 1,624 to 30,144 platelets per cubic millimetre are noted in fatal cases. In mild cases the platelets increase gradually after the febrile period until the normal count is reached or exceeded in the period of convalescence. In tetanus alone the reverse process holds. In all the infections studied — pneumonia, typhoid fever, influenza, erysipelas *et cetera* — the coagulation time of the blood varied inversely to the number of platelets per cubic millimetre; for example, with 55,000 platelets the coagulation time was four minutes twenty seconds; with 624,000 platelets it was forty seconds. These figures were observed in a case of pneumonia, during the illness. Taking pneumonia as a type of infectious diseases, on the fourth to the sixth day of the disease about 100,000 platelets were observed per cubic millimetre. On the day of the crisis, or the day before, the number rapidly increased to 300,000. This number increased still further for four days, up to 624,000 per cubic millimetre, and then began to decline.

"Antuitrin-S."

H. KOPLIN (*The Journal of the American Medical Association*, February 1, 1936) records glycosuria caused by administration of "Antuitrin-S". Because favourable results had been reported, the author treated a boy aged thirty months with bilateral undescended testes by giving an injection of one cubic centimetre of "Antuitrin-S" on three days a week for eight weeks. Enuresis and severe polydipsia occurred, and well-marked glycosuria was noted, which persisted for three weeks after the "Antuitrin-S" was discontinued. The boy returned to normal health, but the testes remained undescended.

British Medical Association News.

MEETING OF THE FEDERAL COUNCIL.

A MEETING of the Federal Council of the British Medical Association in Australia was held at the British Medical Association House, 135, Macquarie Street, Sydney, on August 24 and 25, 1936, SIR HENRY NEWLAND, the President, in the chair.

Representatives.

The following representatives of the Branches were present:

New South Wales: Dr. J. Adam Dick, C.M.G., Dr. George Bell, O.B.E.
Queensland: Dr. D. Gifford Croll, C.B.E., Dr. T. A. Price.
South Australia: Sir Henry Newland, C.B.E., D.S.O., Dr. Bronte Smeaton.
Tasmania: Dr. W. E. L. H. Crowther, D.S.O.
Victoria: Dr. J. Newman Morris, Dr. F. L. Davies.
Western Australia: Dr. D. D. Paton.

Minutes.

The minutes of the previous meeting of the Federal Council, on March 16, 1936, which had been circulated among members, were taken as read and signed as correct.

Finance.

The financial statements and balance sheets for the twelve months ended June 30, 1936, of the Federal Council and of the Australasian Medical Congress (British Medical Association) Fund were presented by the Honorary Treasurer, Dr. George Bell, and received.

The Honorary Treasurer reported that an objection lodged in regard to Federal income tax had been disallowed by the Commissioner, but that he had received a refund in connexion with State income tax for the Congress Fund.

Reference was made to the grant of £1,000 that had been made by the Council of the Parent Body to the Federal Council, and Dr. George Bell, acting on behalf of Dr. J. G. Hunter, the General Secretary, who was absent on leave, read a notice of motion that had been received from Sir Henry Newland, as follows:

> That the grant of £1,000 received from the British Medical Council in London be apportioned to the Branches of the British Medical Association in Australia on the basis of 4s. 6d. per each effective member as at June 30, 1936.

Sir Henry Newland explained his views on the subject. He pointed out that the discussion which led to the grant had originated in a desire on the part of members of some of the Branches to have their subscription to the Association reduced. He also traced briefly the conversations that he and Dr. Newman Morris had had in England with the Home Council. He said that he did not intend that any action on the lines indicated in his motion should be taken without reference to the Parent Body.

Dr. J. Newman Morris said that the Victorian Branch was concerned with the organization of the Federal Council and not with the easing of individual burdens of members. He did not think that there was ever any idea of returning anything to members. He also referred to the terms in which the grant was made. He thought that if Sir Henry Newland's motion were carried it should be submitted to the Home Council before it was put into effect.

Dr. Bronte Smeaton spoke in favour of the motion.

Dr. W. E. L. H. Crowther said that the Tasmanian Branch held that the grant was intended for, and should be made available to, the Federal Council. His Branch Council thought that the activities of the Federal Council should be extended.

Dr. D. Gifford Croll endorsed Dr. Newman Morris's remarks. The money, he said, was intended for the Federal Council. At present the activities of the Council were not very extensive, but if all the Australian Branches were in the position in which the Queensland Branch found itself, much more money would be required for use by the Federal Council.

Dr. D. D. Paton thought that the money should be used entirely for the secretariat of the Federal Council.

At this stage Dr. Bell read replies that had been received from the Branches in reference to the notice of motion. The Western Australian Branch thought that the grant should be retained by the Federal Council. The South Australian Branch left the matter in the hands of its delegates. The Tasmanian Branch was opposed to the motion. The New South Wales Branch thought that the whole of the grant should be used by the Federal Council. The Victorian Branch thought that the money should be applied as the Home Association had intended—for the purposes of a federal secretariat.

The motion was put to the meeting and lost.

Sir Henry Newland said that the Council had next to determine how the money would be spent.

Dr. George Bell reported that the New South Wales Branch Council was of the opinion that the Federal Council should meet occasionally in States other than New South Wales and Victoria. It was thought, for example, that the hands of the Queensland Branch might be strengthened if the Federal Council were to meet in Brisbane.

Dr. D. Gifford Croll said that one objection to the holding of Federal Council meetings in other States was that there would be little likelihood of the meetings being fully attended. He thought that, as the subject of national insurance was likely to arise in the near future, someone should be retained to study the matter and to watch its developments in other countries.

Dr. W. E. L. H. Crowther thought that a first-class man should be retained by the Federal Council to gain knowledge on the subject of national insurance.

After further discussion the Council carried a motion, moved by Dr. Newman Morris, to the effect that consideration should be given to the appointment of a full-time Federal Medical Secretary and that a subcommittee should consider the probable cost of such an appointment and also the cost of a small executive of the Federal Council. It was resolved that the subcommittee should consist of Dr. F. L. Davies and Dr. J. Newman Morris.

Medical Officers' Relief Fund (Federal).

Dr. George Bell presented a report of the Trustees of the Medical Officers' Relief Fund (Federal). The report was received.

Annual Report of the Federal Council for Transmission to London.

Dr. George Bell presented the draft of a report of the activities of the Federal Council for the twelve months ended June 30, 1936, for transmission to the Council of the Parent Body in London and to the councils of the Australian Branches. The report was approved.

The Seventh Australian Cancer Conference.

A letter was read from the Director-General of Health, Commonwealth Department of Health, in which the Director-General expressed his appreciation of the cooperation between the Federal Council and the Branches of the British Medical Association in connexion with the Seventh Australian Cancer Conference. The letter was received.

Reciprocity between State Hospitals in Different States.

Dr. George Bell read a letter addressed to Sir Henry Newland regarding the admission to State hospitals of patients from other States. In this letter it was pointed out that a patient suffering from tuberculosis had come from Wilcannia to Adelaide. The patient had made

certain progress under treatment and her medical attendant had sought her admission into a State institution in Adelaide that was intended for the treatment of such patients. Admission had been refused on the ground that the patient came from New South Wales. The patient was indigent and as a result of this decision had suffered hardship. Sir Henry Newland thought that reciprocity should be established between State institutions of the several States.

Dr. J. Newman Morris pointed out that similar hardships were suffered by patients who lived in towns adjoining the border of a neighbouring State and who were taken ill while they were in that State.

It was resolved that the matter should be referred to the Director-General of Health of the Commonwealth Department of Health with a request that he would bring it before the next meeting of the Federal Health Council.

Formation of Special Associations within the Profession.

At the previous meeting of the Federal Council consideration was given to the formation of special associations within the profession. This had been the subject of discussion between representatives of the Home Association and the Federal Council at the time of the 103rd Annual Meeting of the Association in Melbourne in September, 1935. At the last meeting of the Federal Council it was resolved that a subcommittee should be appointed to draw up a regulation for the formation of special groups or sections within the British Medical Association in Australia and that it should also draft the necessary alterations to the Articles of Association and By-Laws of the Federal Council. Dr. George Bell and Dr. J. Adam Dick were appointed to act as this subcommittee. Dr. George Bell reported that the subcommittee had no report to offer, but presented some extracts of a letter received from England from Dr. J. G. Hunter, General Secretary of the Federal Council. In this letter Dr. Hunter reported that he had conferred with Dr. G. C. Anderson, the Medical Secretary of the Association; and he expressed the view that, as far as the Federal Council was concerned, all that was required was the addition of a new article and by-laws. The new article referred to by Dr. Hunter was as follows:

> Special groups of members having distinctive professional interests may from time to time be formed and dissolved in such circumstances and manner and shall have such status and privileges as may be prescribed or determined by or in accordance with the by-laws.

It was resolved that this new article should be approved and that the subcommittee should draft by-laws in accordance with the article for submission to the several Branches before the next meeting of the Federal Council.

Australasian Medical Congress (British Medical Association).

The Honorary Secretary, Dr. George Bell, reported that an advance of £100 had been made to the executive of the Fifth Session of the Australasian Medical Congress (British Medical Association) towards the initial expenses.

Copies of the minutes of the meetings of the Executive Committee of the Fifth Session were received.

According to congress regulations, each Branch has the privilege of nominating one of its members to be vice-president of each session. Communications were received from several of the Branches, which nominated vice-presidents as follow: The New South Wales Branch nominated Dr. A. A. Palmer, the South Australian Branch nominated Dr. H. Swift, the Tasmanian Branch nominated Dr. G. E. Clemons, and the Victorian Branch nominated Dr. J. Newman Morris. These nominations were accepted and the appointments made accordingly.

The Ethical Rules of an Unincorporated Branch.

A communication was received from the Victorian Branch regarding the ethical rules of an unincorporated Branch. It was pointed out that peculiar circumstances had led to the consideration of this matter by the Branch. Dr. J. Newman Morris thought that the other Branches should be asked whether in their opinion any alterations or additions should be made to the ethical rules which were issued in 1914. It was resolved that this action should be taken.

Repatriation Department.

A letter was received from a member of the Victorian Branch asking the Federal Council to consider the procedure of the Entitlement Appeals Tribunal of the Department of Repatriation. It was held that in some instances a miscarriage of justice might take place.

Dr. W. E. L. H. Crowther said that in his experience in Tasmania the Entitlement Appeals Tribunal was an effective body that did good and useful work.

The Royal Australian Naval Service.

In many previous meetings of the Federal Council the conditions of service of medical officers in the Royal Australian Navy had been considered and the Federal Council had made efforts to improve conditions so that they would be comparable with those obtaining in the Royal Navy. It was reported that a communication had been received from the Navy Office setting out the conditions of entry for short service commissions in the Royal Australian Navy. These commissions were for either three or five years. The rate of pay was £492 *per annum*, rising to £526 *per annum* after three years' service. In addition, subject to certain stipulations, a gratuity of £400 was paid after three years' service and of £1,000 after five years' service. The conditions were considered in detail and the communication was received.

Amidopyrin.

A letter was received from the Victorian Branch requesting that representation should be made to have amidopyrin included in the list of "Specified Drugs" in all States. Dr. Bell reported that the views of the Victorian Branch had been submitted to the other Branches. The Victorian view was supported by Tasmania, Western Australia and New South Wales. The South Australian Branch stated that the draft of the new Act in that State included amidopyrin in the suggested category.

In discussion it was suggested that the several Branches should approach the health authorities in their own States. Eventually the suggestion of Sir Henry Newland was adopted, that the Director-General of Health of the Commonwealth Department of Health should be asked to use his influence to have the suggestion of the Victorian Branch carried into effect.

Recruitment of Members of the Medical Profession.

A letter was received from the Western Australian Branch in which a scheme was suggested for the safeguarding of the interests of medical practitioners who were required to leave their practices in the event of mobilization in war time. Attention was drawn to a minute adopted by the Federal Committee in February, 1918, and a copy of the minute was circulated amongst members. It was resolved that the minutes of the Federal Committee of February, 1918, should be circulated among the Branches for their comment.

The Training of Medical Students in the Combating of Gas Warfare.

A communication was received from the New South Wales Branch in which it was stated that the Branch Council had suggested that medical students should during their undergraduate training receive special tuition in the treatment of wounds and disabilities arising from the use of phosphorus, thermit and poison gas.

This suggestion had been made to the Faculty of Medicine of the University of Sydney. A reply had been received from Professor H. R. Dew, Dean of the Faculty, to the effect that disabilities of the kind mentioned were not part of civil life and that tuition in their treatment would be rather redundant. Professor Dew had suggested

that the Federal Council should approach the Minister for Defence with the request that he should appoint a committee to prepare a brochure dealing with the whole subject. Dr. J. Adam Dick and Dr. D. Gifford Croll moved that the Federal Council should act upon Professor Dew's suggestion. Dr. J. Newman Morris pointed out that the subject of gas warfare had recently been discussed at a meeting of the Victorian Branch and that the meeting had been attended by officers of the Department of Defence. Dr. W. E. L. H. Crowther and Dr. F. L. Davies moved that each Branch should form a committee to consider the measures that should be taken to combat gas attacks. Dr. Crowther and Dr. Davies's motion was carried.

Civil Aviation Landing Grounds.

A communication was received from the New South Wales Branch suggesting that the Federal authorities should be approached in regard to the provision of civil aviation landing grounds, particularly in the country. The matter had first been brought forward by a New South Wales surgeon, who reported that he and other medical practitioners had had unpleasant experiences in attempting to find landing grounds when summoned to attend patients by aeroplane in the country. The New South Wales Branch had written to the State Minister and the Minister had replied that the matter was one purely of finance and that the provision of landing grounds lay entirely with the municipalities.

Dr. F. L. Davies said that there were other problems of far greater importance than the one under discussion, for instance, that of sanitation in country towns *et cetera*. He thought that attention should be devoted first of all to the greater needs.

Dr. W. E. L. H. Crowther thought that the cooperation of the Minister of Defence should be sought.

Dr. J. Newman Morris said that in Victoria the body that should be approached was the Municipal Association.

It was resolved that the Federal Council should communicate with the municipal associations in the several States and that the Branches should be asked to bring the matter before the authorities in their own States.

National Health and Medical Research Council.

Consideration was given to the proposal of the Commonwealth Government to establish a National Health and Medical Research Council. The subject was introduced by consideration of a resolution of the Seventh Australian Cancer Conference that had been moved by Sir Henry Newland. The resolution was as follows:

> This conference recommends that the Commonwealth Government, with the cooperation of the State Governments, form a National Medical Research Council, on which there shall be: (i) representatives of the Commonwealth and State Health Departments; (ii) representatives of the Universities, British Medical Association, Royal Australasian College of Surgeons and other appropriate bodies.

Sir Henry Newland explained briefly the reasons why he had moved the motion at the Cancer Conference. He recognized that the constitution of the proposed body was not the same as had been suggested by the Royal Commission on Health of 1925 and by the Federal Committee at a later date. He thought, however, that if the present proposal were adopted a Medical Research Council such as was desired would eventually be evolved. In any case, half a loaf was better than no bread.

A letter was read from the Victorian Branch expressing the opinion that the proposed National Health and Medical Research Council should not replace the present Federal Health Council, but that the Medical Research Council should be a separate body with separate functions.

Dr. Bell reported that a common letter had been written to the Branches. Tasmania had supported the Victorian Branch view. The South Australian Branch had left the matter in the hands of its delegates. Both the Western Australian Branch and the New South Wales Branch held that the Federal Health Council and the Medical Research Council should be separate bodies, and the Queensland Branch endorsed the opinion expressed by the Victorian Branch.

Dr. J. Newman Morris agreed that the Federal Council should reaffirm its belief that the Medical Research Council should be a separate body. At the same time he held that the Federal Council should be prepared to cooperate with any Commonwealth body that was established to further research.

After discussion, in which every member of the Council took part, it was eventually resolved:

> That while this Council reaffirms its belief that a Medical Research Council should be a separate body, it is willing to cooperate with the proposed National Health and Medical Research Council in the belief that it will in the future be a means of uniting the States and the Commonwealth in medical research. It expresses the opinion that the proposed Council could function as regards research only through an executive composed chiefly of research workers, and it desires to impress the importance of following as closely as possible the Council's recommendations contained in its letter of April 4, 1935.

The Council's letter of April 4, 1935, set out the view of the Federal Council in regard to the constitution of any proposed Medical Research Council. It was resolved that a copy of the resolution should be sent to the Commonwealth Department of Health and that it should be accompanied by a copy of the letter of April 4, 1935.

A Federal Policy for General Medical Services.

A communication was received from the Queensland Branch stating that it was desirable that a Federal policy for general medical services should be laid down. Dr. Bell reported that a common letter had been written to the Branches and that replies had been received from New South Wales and Tasmania. The Tasmanian Branch supported the suggestion for a federal policy, but did not consider its immediate operation advisable, and the New South Wales Branch agreed with the proposal of the Queensland Branch.

Dr. T. A. Price pointed out that the subject had been continuously before the Federal Council and the Federal Committee for a period of twelve years. He was strongly of opinion that the time had come when general principles should be formulated governing the establishment of a State or Commonwealth medical service. The matter had been discussed by the Federal Council the year previously, and Dr. Price thought that the principles put forward on that occasion should be adopted. On his motion the following principles were adopted:

1. That a Commonwealth Insurance Department be established under the Minister of Health for the insurance of all below a certain income limit against all sickness, the practising profession to be adequately represented by nominees in the administration.

2. That the medical services to the community be based on the provision for every individual of a general practitioner or family doctor.

3. The services should be complete. They could cover in addition a general practitioner service, the provision of specialist, ancillary, maternity, pharmaceutical, dental and institutional services.

4. There should be free choice of doctor.

5. That payment of the general practitioners be by capitation fees.

6. That payment for special services be by partial or full payment by the department for each service rendered.

7. That as regards the control of the purely professional side of the services the guaranteeing of the quality of the service and the disciplining of the doctors taking part in it, as much responsibility as possible should be placed on the organizing medical professor.

Federal Medical Registration.

The Council had before it a notice of motion by Dr. J. Newman Morris on the subject of uniform medical registration for Australia. The notice of motion had been communicated to the Branches, and among the replies was a long and detailed report submitted to the Western Australian Branch by Dr. D. D. Paton and Dr. D. M. McWhae. Reference was also made to a report on uniform medical registration that had been considered by the Federal Committee in 1918. After discussion it was resolved:

> That this Council is of opinion that the various State medical Acts should be uniform in drafting and that a committee consisting of Dr. D. D. Paton and Dr. J. Newman Morris, with the President, be requested to report on the best means by which this could be brought about.

Broadcasting Health Talks.

Further consideration was given to the control of the broadcasting of health talks throughout the Commonwealth. At previous meetings of the Federal Council it had been reported that the Minister of Health, on being approached, had replied that it would be necessary for the Federal Council to give specific instances in which it was thought that harm had been done. At the previous meeting of the Federal Council it was resolved that members of the Branches should be urged to be watchful and to report to their Branch Councils instances in which misleading information was given and in which harm resulted. Dr. Bell said that reports had been received from several members, and read extracts from these reports.

Catgut.

At the previous meeting of the Federal Council consideration was given to the manufacture of catgut, and the Branches were asked to bring the matter before the health departments in the several States. The action undertaken by the Branches was reported, and on the suggestion of Dr. J. Newman Morris it was resolved that the whole subject be referred to the Federal Health Council.

Time and Place of the Next Meeting.

The determination of the time and place of the next meeting was left in the hands of the President.

Votes of Thanks.

A vote of thanks was accorded to the Council of the New South Wales Branch for having provided accommodation for the meeting and for its hospitality, and to Sir Henry Newland for the manner in which he had conducted the business of the meeting.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held at the Royal North Shore Hospital of Sydney on June 18, 1936. The meeting took the form of a series of clinical demonstrations by members of the honorary staff.

Asthma.

DR. F. GUY GRIFFITHS showed a male patient, aged twenty-two years, who had been admitted to hospital on January 10, 1936, suffering from asthma. The patient's grandmother had suffered from hay fever and the patient had lived at McMahon's Point, Sydney, all his life. He had been an invalid pensioner since the age of nineteen. The patient suffered from osteomyelitis eleven years ago and had four operations performed on this account. He was first admitted to the Royal North Shore Hospital of Sydney in 1925, at the age of ten years. At this time he was suffering from asthma following an acute bronchitis. He had had asthma for two years before that date. At that time he also had chronic tonsillitis, enlarged post-nasal adenoids and a deflected nasal septum, for which he was operated on successfully. For a while he obtained relief from calcium chloride given in syrup and water.

In June, 1925, he returned to the hospital and was seen by several members of the staff. Calcium chloride was again effective, but in September of that year his condition relapsed and responded to a compound lobelia mixture and to injections of adrenaline. He was tested with albumose-free tuberculin, but gave no reaction. When adrenaline failed, pituitrin was found to be effective. In February, 1926, and in April, 1929, he had further attacks. He was readmitted to hospital in 1933 and was at first relieved by ephedrine. Then when, as usual, the effect of the new drug failed, he obtained relief from pseudo-ephedrine. Shortly after this he became an invalid pensioner, as he was thought to be totally and permanently disabled. In 1935 the patient was twice admitted to hospital. Dr. Griffiths explained that Dr. Blashki had examined the patient and had advised against further operation, because in his opinion the radiological appearance of the maxillary sinus suggested the result rather than the cause of asthma.

Dr. Griffiths gave details of the findings on physical examination of the patient and pointed out that he had been found to be sensitive to horse dander, feathers, cat hair, dust, egg white, pork, codfish and potato. He also set out in tabular form the number of attacks of asthma which the patient had had in each of twenty-two consecutive weeks. The largest number of attacks in any one week was seventy-two and the smallest was three. Recent attacks had been less severe while the patient was taking 2·4 grammes (40 grains) of sodium mandelate four times a day and 0·6 gramme (10 grains) of ammonium chloride six times a day. This had reduced the pH of the urine from 6·8 to 5·12.

Hemiplegia Following Pneumonia.

Dr. Griffiths also showed a child who was admitted to hospital on November 10, 1935, at the age of eleven months, with a history of having had a cold for one week, with feverishness and cough, followed by convulsions. The child had had diphtheria five months earlier.

At the time of admission to hospital the child had had a convulsive seizure, mostly on the right side. During the convulsive attack the child became blue, râles were audible in the chest, and he looked very ill. His temperature was 38·3° C. (101° F.), his pulse rate was 150 and his respiratory rate 40 in the minute. The percussion note was normal, but multiple rhonchi were audible in both lungs and the breath sounds were obscure. The heart appeared clear. The convulsions recurred three or four times. After an action of the bowels the convulsions ceased and transient nystagmus appeared with paralysis of the right arm and leg and a Babinski sign on the right side. On the following day the child became desperately ill. The pulse rate was 180 and the respiratory rate 76 in the minute, and the temperature was 39·4° C. (103° F.). The child was pale and restless and completely paralysed in the right arm and leg. A strong Babinski reflex was present on the right side, but not on the left. Scattered patches of dulness appeared in both lungs, with high-pitched breath sounds. Two lumbar punctures were performed under chloroform anæsthesia, but no cerebro-spinal fluid was obtained. The temperature became normal after four days and the child began to look well. The hemiplegia on the right side was complete for three or four days and then the paralysis began to disappear. No trace of weakness was discoverable when the child was discharged from hospital on November 23, 1935.

Dr. Griffiths explained that treatment had consisted in the rectal administration of 0·6 gramme (10 grains) of bromide of potash and chloral hydrate. One hypodermic injection of 1·0 milligramme (one-sixtieth of a grain) of morphine was given, and this was followed by oil and by *Hydrargyrum cum Creta*, 0·015 gramme (one-quarter of a grain) three times a day. The temporary hemiplegia was

probably due to a meningeal hæmorrhage resulting from coughing, as sometimes occurred in whooping cough. At the time of the demonstration the child could walk fairly well and was otherwise very well.

Pseudo-Hypertrophic Muscular Atrophy.

DR. C. W. SINCLAIR showed a boy, aged eleven years, who was suffering from pseudo-hypertrophic muscular atrophy. Weakness had been noticed for at least five years. The condition had not advanced during the last year. The boy had the characteristic early waddling gait, and experienced some difficulty in climbing high steps. Lordosis was noticeable when he stood upright, and he had hypertrophy of the muscles of the calves and forearms and also of the glutei. There was wasting with loss of power in practically all the other muscles, and this was particularly noticeable in the muscles of the shoulder girdle. The child had been treated with glycine for the last eighteen months.

Tumour of the Anterior Thoracic Wall.

DR. F. J. BRIDGES showed a male patient, aged seventy-two years, who was suffering from a tumour of the anterior thoracic wall. The patient's previous illnesses included an injury to the left eye fifty years ago, *herpes zoster* twenty years ago, and coronary sclerosis ten years ago. Six years ago the patient was submitted to the operation of prostatectomy.

Nine months ago the patient first noticed a lump on the left side of the chest; it was slightly painful and was growing slowly. He gave a history of breathlessness on exertion for ten years. Physical examination revealed two lumps on the chest over the second and third ribs. The medial of the two masses fluctuated and was tender, but was not connected with bone or skin. It was apparently connected with the *pectoralis major* muscle. The lateral mass was larger and not fluctuant; it was not tender. The patient was apyrexial. The heart was dilated and somewhat hypertrophied. The systolic blood pressure was 160 and the diastolic pressure 90 millimetres of mercury. Examination revealed great thickening of the pleura over the left side of the chest. Radiologists suggested that only a slight amount of fluid was present in the pleura. No involvement of the upper ribs on the left side was detected radiologically. Both masses were aspirated. Fluid from the medial lump contained tubercle bacilli.

Primary Pulmonary Tuberculosis.

DR. COTTER HARVEY AND DR. HAROLD WILSON demonstrated two children suffering from primary tuberculous infection of the lungs.

The first patient, a girl, aged eleven years, was first seen in 1927 at the age of two and a half years as a contact of her father. At this time the von Pirquet test gave no reaction and X ray examination of her chest revealed no abnormality. She was examined every year, skin tests, both cutaneous and intracutaneous (Mantoux), giving no reaction until 1935, the year in which the father died. In February of this year the child gave a positive response to the Mantoux test, and X ray examination of her chest revealed an exudative patch in the right lung beneath the clavicle; the hilar glands were enlarged. Serial skiagrams were shown from this time up to the present, illustrating the changes in appearance of the primary complex. The lesion was now progressing satisfactorily towards healing, and there was radiological evidence of calcification in the hilar lymph glands. It was pointed out that the child's health during the whole of this period had remained good, and there had been no symptoms or clinical manifestation of any active disease. This was the type of case found only during routine examination of contacts or examination of large series of school children.

The second patient was a boy of three and a half years, also a contact of his father, whose skiagram twelve months previously had revealed no abnormality. He now presented radiologically the typical picture of a primary complex in the upper lobe of the left lung, with a positive reaction to the Mantoux test. His only symptom was failure to gain in weight, as there had been an increase of only 0·45 kilogram (one pound) in the previous twelve months.

Lymphadenoma of the Lung.

Dr. Cotter Harvey and Dr. Wilson also showed a patient suffering from lymphadenoma of the lung, which was responding to X ray therapy. It is hoped to report this case in full at a later date.

A Tipping Frame for Use in Bronchiectasis.

Dr. Cotter Harvey and Dr. Wilson next demonstrated a special tipping frame for use in the postural drainage of patients suffering from bronchiectasis. It was explained that this frame was copied from one used in the Alfred Hospital, Melbourne.

Arsenical Neuritis and Pulmonary Neoplasm.

Dr. Cotter Harvey and Dr. Wilson's next patient had been admitted to a country hospital seven months previously, suffering from influenza. Soon after his admission it was noted that he had pains in his arms and legs, and he gradually lost the use of all his limbs. As his nervous symptoms had not improved he was transferred to the Royal North Shore Hospital under the care of Dr. Stuart Scougall for orthopædic treatment. It was proven by examination of his hair that his symptoms were due to arsenical neuritis. The source from which he had presumably ingested arsenic for a considerable period of time had not yet been ascertained. Three weeks before the demonstration the patient had had an hæmoptysis, and though no definite signs were found in his chest, X ray examination revealed a shadow extending out from the right hilar region to the lower lobe, which was strongly suggestive of neoplasm. It was considered likely that this had been present at the time of his first illness and that there was no relation between the lung condition and the nervous disorder.

Lead Poisoning.

DR. W. W. INGRAM showed a male patient, aged twenty-nine years, who was suffering from lead poisoning. The patient's illness began in 1933, when, after he had been working for about three months in lead processes in the manufacture of motor car batteries, he noticed a feeling of numbness, combined with gradually increasing weakness and wasting in the legs. He said that he had always taken great care in washing his hands before meals. Soon he could not walk far without suffering from severe cramps. About this time he complained of severe colicky pains in his abdomen, and his bowels were very constipated. His tongue was continually coated and he had a foul taste in his mouth. He had lost 37·8 kilograms (three stone) in weight in about four months. He became very pale and suffered from frequent attacks of faintness and giddiness. Any slight exertion made him breathless. He suffered greatly from headaches, and any bright lights used to worry him considerably.

Dr. Ingram pointed out that the patient had been under continuous treatment at various metropolitan hospitals since 1933. His weight had increased and the weakness in his legs and his abdominal symptoms had disappeared. At the same time, although he had not been in contact with lead since 1933, his anæmia had not improved and he still showed a typical picture of lead poisoning. The remains of a blue line were present in the gum margins of the incisor teeth. His mucous membranes were pale and he was colourless. Some tenderness was noted on palpation of the left side of the abdomen, but no other abnormality was present in any of the other systems. Blood examination revealed a total red cell count of 4,800,000 per cubic millimetre, the hæmoglobin value was 60% and the colour index 0·63. Anisocytosis and poikilocytosis were present; many of the red cells showed polychromasia and punctate basophilia was present in 1·7% of the red cells. The total leucocyte count was 6,000 per cubic millimetre, and the eosinophile cell percentage was 1·5.

Diabetes Mellitus.

Dr. Ingram also showed a female patient, aged fifty-seven years, who was suffering from *diabetes mellitus* and who had been under observation at the diabetic clinic since 1929. Dr. Ingram said that he was showing the patient to illustrate the effect of a staphylococcal infection on the carbohydrate tolerance. Throughout her clinical history there were found frequent references to her inability to adhere either to a stabilization diet or to her insulin dosage.

The patient was first admitted to hospital in 1931. She was admitted for stabilization and was discharged on a 1,500 calorie diet and taking 40 units of insulin every day. In December, 1934, she was admitted to hospital a second time, suffering from a labial abscess, and she was discharged on a diet of 1,100 calories and taking 40 units of insulin a day. In June, 1935, she suffered from an infected wound of the thumb, and when she was discharged from hospital her diet had been increased to 1,500 calories and her insulin dosage remained the same. After her discharge on that occasion she attended the diabetic clinic more or less regularly until January, 1936, which proved to be her last appearance. She stated, however, that she had adhered to her diet and insulin, but Dr. Ingram pointed out that her statements were unreliable.

The patient's present illness, Dr. Ingram explained, commenced on April 22, 1936. On this occasion she had a large carbuncle in the interscapular region, measuring 15 by 10 centimetres. The carbuncle was treated for several days by orthodox measures at the outdoor department. As her condition did not improve, the patient was admitted to hospital and her urine was found to contain a considerable amount of sugar, acetone and diacetic acid. She was at first put on a diet of 1,100 calories. Her carbuncle was treated by orthodox methods, but in addition she was given one cubic centimetre of manganese every day and her insulin was increased in rapid stages until she was taking 100 units a day. At the same time she was given the routine 1,700 calorie antiketogenic diet. The carbuncle rapidly healed, and after she had been in hospital for eleven days very little discharge was present. At this time the sugar commenced to disappear from the urine and the insulin was rapidly reduced. As her urine still remained sugar-free, it was found that insulin could be suspended altogether. Dr. Ingram showed a table in which the increase in insulin dosage was clearly set out. He pointed out that the patient was now taking a 1,500 calorie high carbohydrate diet and was having no insulin. Her blood sugar at the time of her admission to hospital was 0·25 milligramme *per centum*. At the time of the meeting it was 0·20 *per centum*.

Agranulocytosis.

DR. OSSIAN ROBERTSON showed a woman, a *primipara* aged twenty-three years, who had been admitted to hospital on April 21, 1936. The patient gave a history that her last menstrual period had occurred on August 20, 1935. She was quite well during her pregnancy, until April 21, 1936, when, on attending the ante-natal clinic, she was found to have œdema of the legs and albuminuria. Her systolic blood pressure was 100 and her diastolic pressure 60 millimetres of mercury. Ulcers were present on the tongue and the buccal mucous membrane. The patient's temperature was 38·9° C. (102° F.). No symptoms of preeclampsia were present, but the patient looked very ill. She gave a history of having had measles and diphtheria as a child, but there was no history of her having taken amidopyrine or allied compounds.

At the time of her admission to hospital the patient complained of frontal headache, epigastric pain and pain in the right side of the chest, which became worse on respiration. The patient vomited several times and had a slight epistaxis. Dr. Robertson described her signs and symptoms at this time and said that the provisional diagnosis was preeclamptic toxæmia and right lobar pneumonia.

On April 23, 1936, the patient complained of a sore throat. On examination her fauces were injected and the right tonsil was enlarged and covered with a white membrane. Cervical lymphadenitis was present. The patient's temperature was 38·3° C. (101° F.), her pulse rate was 120 and her systolic blood pressure was 110 millimetres of mercury. The urine contained "one-tenth albumin". A small pleural effusion was present at the base of the right lung. In smears taken from the throat neither the organism of Vincent's angina nor the Klebs-Löffler bacillus was found. Subsequently the condition of the throat and mouth improved and the patient's temperature fell to 37·2° C. (99° F.).

On May 3, 1936, after a labour lasting seven hours and thirty-six minutes, the patient was delivered of twins. Slight *post partum* hæmorrhage occurred.

On May 11, 1936, the patient became extremely pale, the alveolar mucous membrane was swollen. Ulcers were present in the mouth, but the throat was clear. Cervical lymphadenitis was present with slight abdominal distension. The patient's temperature rose to 41·1° C. (106° F.) and her pulse rate to 140. The urine was clear. A full blood count revealed the presence of agranulocytosis. The red cells numbered 2,200,000 per cubic millimetre, the hæmoglobin value was 26% and the colour index 0·59. The leucocytes numbered 1,200 per cubic millimetre, and of these 91% were lymphocytes and 9% monocytes. On May 16, 1936, the patient was given a blood transfusion and both "Edwenil" and "Campolon" were given. Recovery was uneventful, and by the end of May the patient was up and walking about.

Concealed Accidental Hæmorrhage.

Dr. Ossian Robertson also showed a woman, aged twenty-nine years, a *multipara*, who had suffered from concealed accidental hæmorrhage. The patient had albuminuria during her first pregnancy, the second being normal. Labour was expected to occur on February 20, 1936, and everything was normal until January 14, when the patient complained of slight œdema of the feet and ankles and a cloud of albumin was found on examination of the urine. The systolic blood pressure was 130 and the diastolic pressure 80 millimetres of mercury. The patient was put on a diet, and on February 11 merely a trace of albumin was found in the urine. On February 12 the patient came into hospital complaining of slight hæmorrhage and of pain in the abdomen of five hours' duration. On examination she looked very pale. Her pulse rate was 110 per minute. No pronounced abdominal tenderness or rigidity was present. Irregular uterine contractions were occurring and slight vaginal hæmorrhage was noted. The urine contained "one-quarter albumin". For the next two hours irregular pain continued, but no vaginal hæmorrhage occurred. The pulse rate remained the same, but the patient looked extremely pale. Three hours after her admission to hospital vaginal examination showed that the *os uteri* was dilated "three fingers". The membranes were artificially ruptured and a large quantity of blood-stained *liquor amnii* escaped. Cæsarean section was immediately performed.

When the abdomen was opened the peritoneal cavity was found to be full of blood, which was leaking out through the tubes. The blood had escaped through the uterine wall and was distending the broad ligament; the peritoneum on the front of the uterus was distended and was pushing the bladder off the uterus. The uterus was opened and a stillborn fœtus was removed. The patient's recovery was uneventful.

Dr. Robertson drew attention to two interesting points. In the first place the patient showed no signs of shock to correspond with her great blood loss, and in the second place the usual severe abdominal pain and tenseness of the uterus were not present, owing to the fact that the blood ran out of the uterus through the tubes into the peritoneal cavity.

Chronic Empyema.

DR. V. M. COPPLESON showed a woman, aged fifty-seven years, who had suffered from chronic empyema. The patient was admitted to hospital on February 21, 1935, with a provisional diagnosis of lobar pneumonia. She had cough and rusty sputum, and pain had been present in the right side of the chest for fourteen days. Two days

after her admission to hospital X ray examination revealed dulness of the lower third of the right side of the chest, and it was thought that the dulness was caused by encysted fluid. About a fortnight later *paracentesis thoracis* was performed, but no fluid was obtained. Bronchoscopy was undertaken on three occasions and mucus was aspirated from the right lower bronchus. On the third occasion lipiodol was injected and X ray examination revealed an abscess cavity at the base of the right lung.

On May 29, 1935, thoracotomy was performed. A hydatid cyst full of dead daughter cysts was found. The cyst was emptied and drained. On the following day X ray examination revealed a pneumothorax in the upper part of the right side of the chest. On June 5, 1935, a further X ray examination was made. The lung was still considerably collapsed and fluid was still present. On September 25, 1935, thoracoplasty was performed. Portions of three ribs were removed and a cavity containing pus and surrounded by a thick cartilaginous wall was found. As much as possible of the wall of the cavity was removed.

On January 9, 1936, X ray examination showed that improvement was taking place and there was no evidence of the previous cavity. Considerable fibrosis and irregular consolidation of the base of the lung were seen.

On March 25, 1936, thoracotomy was again performed and several fragments of dead bone were removed from the old thoracotomy scar close to the resected rib. On June 1, 1936, the sinus was closed, the wound being almost healed, and very slight discharge was present.

Lymphangioma of the Tongue.

DR. L. S. LOEWENTHAL showed a female patient, aged thirty-one years, who complained of swelling in the throat and of difficulty in swallowing of four to five weeks' duration. The patient said that she had had something wrong with her tongue all her life. When she was three years of age the condition appeared as "white threads on her tongue". The lesion had been scraped and cauterized on different occasions, but it slowly became worse. During the previous three months her neck, underneath the left side of the jaw, began to swell. A blood count revealed the following information:

Erythrocytes, per cubic millimetre ..	4,600,000
Hæmoglobin value	86·0%
Colour index	0·93
Leucocytes, per cubic millimetre	7,000
Neutrophile cells	73·5%
Lymphocytes	20·0%
Monocytes	4·5%
Eosinophile cells	1·5%
Basophile cells	0·5%

The red cells were normal in size, shape and staining.

Neither the Wassermann nor the Kahn test yielded any reaction. Dr. Loewenthal said that a biopsy from the tongue had been made. The section showed a great deal of epithelial hyperplasia to form papillary outgrowths with a core which showed many dilated lymph channels. Some of the channels were filled with coagulated lymph, in some cases mixed with blood. The condition was apparently one of cavernous lymphangioma.

Bronchiectasis Improved by Bronchoscopy.

DR. E. P. BLASHKI showed a male patient, aged fifty-seven years, a hairdresser, who was suffering from bronchiectasis. The patient was admitted to hospital on March 20, 1936. He had been in his usual health until ten weeks prior to his admission, when he thought that he had swallowed a fish bone. Since then he had had a severe cough night and day, and had coughed up daily about three ounces of foul greenish muco-purulent sputum, which was occasionally blood-stained. He had had severe dyspnœa on exertion and frequent night sweats. He had lost 12·6 kilograms (two stone) in weight and his appetite was poor. An X ray examination, undertaken on March 7, 1936, had revealed no abnormality, save chronic bronchitic changes. Physical examination of the chest revealed diminished movement in the region of the left base. The percussion note was impaired over the lower lobe of the left lung and dulness was complete at the base. Vocal fremitus was absent over the left lower lobe. The breath sounds were absent over the left lower lobe, except in the upper part, where they were weak and vesicular, with some fine râles. Vocal resonance was very faint. X ray examination on March 21, 1936, revealed changes suggesting bronchiectasis at both bases. Bronchoscopy was undertaken on several occasions after postural drainage had failed to give any relief. On the first occasion a large amount of foul greenish pus and mucus was evacuated from the left lower lobe. A large amount of granulation tissue was found blocking the left bronchus. The tissue was such as might be found around a foreign body, or it might have been malignant tissue. At the second bronchoscopy a piece of tissue was removed for biopsy and proved to be simple granulation tissue. At the third bronchoscopy the instrument was cast further than on the previous occasion. Very little granulation tissue was found and much less pus was present. No foreign body could be found. At the fourth bronchoscopy there was no evidence of polypoid granulation tissue. At the time of the meeting the patient was much better and was putting on weight. The percussion note was still impaired over the left lower lobe and numerous moist râles were present.

Intestinal Polyposis.

DR. E. D. CLARK showed a boy, aged twelve years, who had been admitted to hospital on December 26, 1935, complaining of abdominal pain and of having passed blood *per rectum* for three days. Examination revealed the presence of a mass in the abdomen. At operation, undertaken immediately, the mass proved to be an intussusception which was reduced. The patient was discharged from hospital on January 11, 1936.

He was readmitted on January 31, 1936, complaining of abdominal pain of two weeks' duration. Operation was undertaken on February 4, 1936. Polypi were found in the small intestine and also some enlarged mesenteric glands. The small intestine was short circuited. The patient was discharged from hospital on March 6, 1936.

He was readmitted to hospital on April 26, 1936, complaining of abdominal pain and of vomiting of thirty-six hours' duration. A provisional diagnosis of intestinal obstruction was made. At operation the redundant loop of bowel, measuring five feet in length, was removed. The patient made an uneventful recovery. The pathological report was as follows:

> Intestinal polyposis; intense inflammatory changes; acute congestion with œdema; proliferation of epithelial cells forming papillomatous projections.

Xanthoma Tuberosum Multiplex.

DR. F. C. FLORANCE showed a patient who was suffering from *xanthoma tuberosum multiplex*. He said that this condition consisted of flat or rounded yellow multiple nodules, which, while usually occurring on the elbows and knees, might be found on any part of the skin and also on the mucosæ. It might also occur in the viscera, on periosteum, in the aorta *et cetera*. The commonest lesion was a pear-shaped nodule, but the condition might occur in confluent plaques which covered large areas.

The condition was found principally in adults who had disorders of the liver and biliary passages. Histological examination revealed fatty dégeneration with xanthoma cells. Treatment consisted of removal of the lesions by desiccation if they were scattered and few; either the high frequency current or carbon dioxide snow might be used. Dr. Florance said that *xanthoma diabeticorum* was differentiated from *xanthoma tuberosum multiplex* by its rapid course, by the inflammatory reaction about the lesions and by the association with diabetes.

Dermatitis Artefacta.

Dr. Florance also showed a patient, aged sixteen years, who was suffering from *dermatitis artefacta*. He said that these lesions usually had a distinctive clear-cut and bizarre appearance. The shape and arrangement were such as were not encountered in any other affection. The lesions

were generally distributed on parts easily reached by the hands and had a tendency to be linear. They were rarely seen on the right hand or arm unless the patient was left-handed. Dr. Florance pointed out that in his patient the high arched palate and the insensitive fauces frequently noticed in patients who suffered from these lesions were pronounced.

A Syphilide Resembling Psoriasis.

Dr. Florance's next patient was suffering from a squamous syphilide simulating psoriasis. He pointed out that, although the lesions were on the extensor surfaces, like psoriasis, they gave a feeling to the palpating fingers of being in the skin rather than on the skin; in other words, induration was present.

Generalized Lichen Planus.

Dr. Florance also showed a patient who was suffering from typical acute and widespread generalized *lichen planus*. He said that the diagnostic papules were small, flat, polygonal glistening facets lying between the natural lines of the skin; they were of a characteristic violet colour.

Lichen Planus Verrucosus.

Dr. Florance showed a patient who was suffering from *lichen planus verrucosus*. He said that this was a term applied to the hypertrophic variety of the disease, which occurred most commonly on the shins. It was the chronic localized type of *lichen planus*. At the edges of the patches small flat-topped polygonal papules might be discovered on careful search.

Epithelíomata Treated by Radium.

Dr. Florance also showed several patients suffering from epithelíomata in various regions, in whom successful treatment by radium had been undertaken.

(To be continued.)

NOMINATIONS AND ELECTIONS.

THE undermentioned has been reelected a member of the New South Wales Branch of the British Medical Association:

Anderson, Edith Elizabeth Alice, M.B., Ch.M., 1920 (Birmingham), 11, Nicholson Street, Burwood.

The undermentioned has been elected a member of the New South Wales Branch of the British Medical Association:

Hooper, Kenneth Hugh, M.B., B.S., 1936 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Correspondence.

NUTRITION.

SIR: I congratulate the Queensland Branch of the British Medical Association for its enterprise in breaking away from outworn traditions and publishing the short but pontifically authoritative brochure "Adequate Nutrition During Pregnancy and Lactation". This is to be particularly admired in that no room is left for fiddling variations that ignorant practitioners may recommend in individual cases; everything is definite, decisive and final. It is good to know that at least one branch of the medical art has been finally removed from the doubts and controversies that have beset it. General dietetics also need no longer be studied; for the pamphlet concludes with the comforting assurance that "husband and family too . . . will feel well and keep healthier if the general principles of this dietary schedule are followed".

It is good to vision the day when the work of the Queensland Branch is complete, our work is reduced to the choosing of the specific pamphlet for each patient, and medicine is at last an exact science.

Yours, etc.,

F. F. PINCUS.

Mount Isa,
Queensland,
August 7, 1936.

THE AFTER-CARE OF THE TUBERCULOUS.

SIR: I have just received a letter from Sir Pendrill Varrier-Jones, of Papworth Village Settlement, informing me of a special conference to be held in London in April of next year on the subject of the after-care of the tuberculous. This Dominions After-Care Conference is being conducted under the auspices of the Overseas League and will be concluded just prior to the Royal coronation. It is desired that there should be some Australian representation at the conference. If any colleagues who are interested in the subject and who are likely to be in England at the time will communicate with me, I shall be happy to give further information.

Yours, etc.,

A. R. SOUTHWOOD.

170, North Terrace,
Adelaide,
September 7, 1936.

SCARLET FEVER AND RUBELLA.

SIR: During the last few weeks many of the patients sent to this hospital with a diagnosis of scarlet fever have been found to be suffering from rubella. May I therefore use your columns to draw attention to the present prevalence of German measles and its importance in the differential diagnosis of scarlet fever, of which it is often a surprisingly good imitation.

Yours, etc.,

HALES WILSON,
Deputy Medical Superintendent.

The Prince Henry Hospital,
Sydney,
September 12, 1936.

Obituary.

JACOB ROSENTHAL.

WE regret to announce the death of Dr. Jacob Rosenthal, which occurred on August 28, 1936, at Elwood, Victoria.

Books Received.

THE PATIENT AND THE WEATHER; Volume I, Part 2, Autonomic Integration, by W. F. Petersen, M.D., with the assistance of M. E. Milliken, S.M.; 1936. Michigan: Edwards Brothers, Incorporated. Imperial 8vo, pp. 811, with illustrations. Price: $9.00 net.

PLAGUE: A MANUAL FOR MEDICAL AND PUBLIC HEALTH WORKERS, by Wu Lien-Teh, M.A., M.D., J. W. H. Chun, M.B., B.C., R. Pollitzer, M.D., and C. Y. Wu, M.B., B.S.; 1936. Shanghai: The National Quarantine Service. Royal 8vo, pp. 580, with illustrations. Price: 15s. net.

IT'S IN YOUR KITCHEN: SIMPLE REMEDIES AND HINTS FOR EVERYONE, by A. B. Parry; 1936. Australia: Angus and Robertson. Crown 8vo, pp. 167. Price: 3s. 6d. net.

SOCIAL HYGIENE TO-DAY, by H. E. Garle; 1936. London: George Allen and Unwin Limited. Demy 8vo, pp. 387. Price: 12s. 6d. net.

Diary for the Month.

SEPT. 22.—New South Wales Branch, B.M.A.: Medical Politics Committee.
SEPT. 23.—Victorian Branch, B.M.A.: Council.
SEPT. 24.—South Australian Branch, B.M.A.: Branch.
SEPT. 24.—New South Wales Branch, B.M.A.: Branch.
SEPT. 25.—Queensland Branch, B.M.A.: Council.
OCT. 1.—South Australian Branch, B.M.A.: Council.
OCT. 2.—Queensland Branch, B.M.A.: Branch.
OCT. 2.—New South Wales Branch, B.M.A.: Annual Meeting of Delegates of Local Associations with the Council.
OCT. 6.—Tasmanian Branch, B.M.A.: Council.
OCT. 6.—New South Wales Branch, B.M.A.: Council.
OCT. 7.—Western Australian Branch, B.M.A.: Council.
OCT. 7.—Victorian Branch, B.M.A.: Branch.
OCT. 9.—Queensland Branch, B.M.A.: Council.
OCT. 12.—New South Wales Branch, B.M.A.: Organization and Science Committee.
OCT. 13.—Tasmanian Branch, B.M.A.: Branch.
OCT. 13.—New South Wales Branch, B.M.A.: Executive and Finance Committee.

Medical Appointments.

Dr. A. B. Barry has been appointed Senior Medical Officer, Department of Mental Hospitals, New South Wales.

* * *

Dr. R. R. Bye, Dr. M. R. Morey and Dr. B. A. Serjeant have been provisionally appointed Senior Medical Officers, Office of the Director-General of Public Health, New South Wales.

* * *

Dr. D. M. Steele, of Port Lincoln, South Australia, has been appointed Medical Referee under the provisions of the *Workmen's Compensation Act*, 1932, of South Australia.

* * *

Dr. W. T. Coyle has been appointed Government Medical Officer at Adaminaby, New South Wales.

* * *

Dr. H. L. Chester has been appointed Medical Officer of Health to the Wyalkatchem Road Board, pursuant to the provisions of the *Health Act*, 1911 *to* 1935, of Western Australia.

* * *

Dr. J. G. Drew has been appointed Metropolitan Medical Officer of Health at Sydney, New South Wales.

* * *

Dr. K. D. Hudson has been appointed Government Medical Officer at Cooma, New South Wales.

* * *

Dr. R. M. Windeyer has been appointed Government Medical Officer at Paterson, New South Wales.

* * *

Dr. M. J. Morris has been appointed Medical Officer of Health to the Merredin Road Board, pursuant to the provisions of the *Health Act*, 1911 *to* 1935, of Western Australia.

* * *

Dr. T. M. Gilbert has been appointed Medical Officer of Health to the Youanmi Local Board of Health, under the provisions of the *Health Act*, 1911 *to* 1935, of Western Australia.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages XX-XXII.

LAUNCESTON PUBLIC HOSPITAL, LAUNCESTON, TASMANIA: Resident Medical Officer.
MATER MISERICORDIÆ PUBLIC HOSPITAL, BRISBANE, QUEENSLAND: Honorary Officers.
PERTH HOSPITAL, PERTH, WESTERN AUSTRALIA: Junior Resident Medical Officers.
STATE PUBLIC SERVICE, BRISBANE, QUEENSLAND: Health Officer.
SYDNEY HOSPITAL, SYDNEY, NEW SOUTH WALES: Director of Pathology Institute.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associate Friendly Societies' Medical Institute. Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY Hospital are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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